NOVEMBER

PROPERTY OF DENTAL LIBRARY UNIVERSITY OF MICHIGAN

THE JOURNAL OF THE AMERICAN DENTAL ASSOCIATION

Medicine and Dentistry—*Mayo*
Cleft Lip and Palate—*Risdon*
Enamel Development—*Von Beust*
Vincent's Infection—*Hardgrove*
Dental Care and Family Budget—*Davis*
Metallography of Gold Nickel Alloys—*Shell*
Gold Foil in Modern Practice—*Johnson*
Malocclusion in Mutilated Cases—*Sippy*
New Method of Filling Root Canals —*Buckley*
Tissue Changes in Treated Teeth—*Moen*
Tissue Changes in Traumatic Occlusion —*Orban*
Trigeminal Neuralgia—*Smith*
Cleft Palate—*Shearer*
Attacking "Acid Factories"—*Pfanstiehl*
Surgical Eradication of Pyorrhea—*Ward*
EDITORIALS
Endowments to Dentistry
Styles for November
BUREAU OF CHEMISTRY
Tartaroff
DENTAL HEALTH EDUCATION
Dental Defects in Employment Examinations —*Sappington*

CONTENTS

Harry W. Nelson, D.D.S. ..... Frontispiece

ORIGINAL COMMUNICATIONS

The Interdependence of Medicine and Dentistry.
By Charles H. Mayo 2011

Treatment of Cleft Lip and Palate.
By Fulton Risdon, M.B., F.A.C.S., D.D.S. 2017

Intrafollicular Enamel Development.
By Theodore B. Von Beust, D.D.S. 2021

Vincent's Infection from the Pathologic and Bacteriologic Point of View.
By T. A. Hardgrove, D.D.S. 2032

Dental Care and the Family Budget.
By Michael M. Davis, Ph.D. 2039

Metallography of Gold Nickel Alloys.
By John S. Shell, B.S. 2046

The Value of Gold Foil in Modern Practice.
By C. N. Johnson, D.D.S. 2051

Consideration of Malocclusion in Mutilated Cases.
By Burne O. Sippy, B.S., D.D.S., M.S. 2057

New Method and Technic of Filling Root Canals.
By J. P. Buckley, Ph.G., D.D.S., F.A.C.D. 2069

Tissue Changes in Treated Teeth of Known History.
By O. H. Moen, D.D.S., M.S.D. 2075

Tissue Changes in Traumatic Occlusion.
By Balint Orban, M.D. 2090

Trigeminal Neuralgia and Its Treatment by Alcoholic Injection.
By Arthur E. Smith, D.D.S., M.D. 2107

Posture at the Dental Chair as Related to the Dentist's Health.
By Arthur H. Nobbs, M.S., D.D.S. 2132

Cleft Palate.
By William L. Shearer, A.B., M.D., D.D.S., F.A.C.S., F.A.C.D. 2135

Attacking "Acid Factories" in the Blind Alleys of the Mouth.
By Carl Pfanstiehl 2141

The Surgical Eradication of Pyorrhea.
By A. W. Ward, D.D.S. 2146

Healing of Tooth Sockets Following Tooth Extraction in Dogs.
By Warren S. Schram, D.D.S. 2156

Treatment to Enlarge Abnormally Small Mouth for Full Denture Prosthesis: Report of Case.
By B. S. Burks, D.D.S. 2158

EDITORIAL DEPARTMENT

Endowments to Dentistry.—Styles for November.—Therapeutically Suggestive Names and the Laity.—Deaths Under Anesthetics.—John A. Stoeckley, D.D.S. (1870-1928) 2159-2166

BUREAU OF CHEMISTRY

Tartaroff: A "Tartar-Remover" 2167

BUREAU OF DENTAL HEALTH EDUCATION

The Incidence of Dental Defects in Two Thousand Four Hundred and Sixty-Nine Employment Examinations.
By C. O. Sappington, M.D., Dr.P.H. 2170

Children's Dentistry from a Public Health, as Well as the Private Practitioner's Standpoint.
By L. W. Morrey, D.D.S. 2174

Dental Hygiene as Taught in the Schools of Waterloo, Iowa.
By Clella McCullough 2180

STATE SOCIETY OFFICERS

Directory of State Society Officers and Dates of Meetings 2185

CURRENT LITERATURE

Extracts and Abstracts 2192

MISCELLANY

Book Reviews 2198
Correspondence 2200
News 2200

Announcements 2201

Library Bureau: Some Package Libraries 2204

ADVERTISING

Index to Advertisements A-59
Classified Section A-52, A-53

The Journal of the American Dental Association, Vol. XV, No. 11. Published by the American Dental Association, 58 East Washington Street, Chicago, Ill. Yearly subscription, $2.50. Entered as second-class matter, May 1, 1928, at the post office at Chicago, Ill., under the act of Aug. 24, 1912. Published monthly.

HARRY W. NELSON, D. D. S., Minneapolis, Minn.
Vice President of the American Dental Association

UNIV. OF MICH.

THE JOURNAL
of the AMERICAN DENTAL ASSOCIATION

Vol. 15 NOVEMBER, 1928 No. 11

Original Communications

THE INTERDEPENDENCE OF MEDICINE AND DENTISTRY*

By CHARLES H. MAYO, Rochester, Minn.

THE dentists and the medical men, once of one cult, are interested in the problem of restoring health, giving comfort to the ailing and preventing disease. Some of you may know that the barber pole dates back to the latter part of the sixteenth century when the barber surgeons associated with general surgeons. The red and white represents blood running down the arm when bleeding was considered a cure for certain diseases.

Herodotus, the historian and contemporary of Hippocrates, described the many divisions of medicine, as is done now, and also mentioned those physicians who took care of the teeth. When the world was 3,000 years younger, man made more use of his teeth in eating. Preserved skulls of many Egyptians showed that good teeth were the rule.

The great age of disease is shown by the caries about the teeth in the jaws of the fossilized remains of the extinct historic animals of millions of years ago. While Hippocrates, Celsus, Galen and others made some remarkably good descriptions of individual diseases which occurred during the few hundred years before and after the birth of Christ, the real beginning of medicine came in 1857 with the first publicly recorded research as to the bacterial cause of a disease, and its proof by Louis Pasteur.

In 1509, Galileo had brought distant objects near with the telescope. Lenses were not arranged to magnify the nearby until the time of the Janssen brothers, in 1590. Leeuwenhoek, nearly a hundred years later, by better grinding methods, developed a useful microscope, which showed cell structure and, as curiosities, moving microbic life.

Yet the great importance of the minute life of the world both for good and evil was not appreciated, or at least was not applied, until Pasteur showed the relationship of special germs to the diseases of plant and animal life.

*Read at the Third General Meeting of the American Dental Association, Minneapolis, Minn., Aug. 22, 1928.

For ages, medical men were educated largely by studying and observing, and by assisting those in active practice. Some of these physicians took greater interest than others in diseases of the mouth and teeth; a few developed great mechanical ability, and dentistry evolved.

In this country, medical schools had existed many years before the first school of dentistry was opened, in Baltimore, in 1840, and this in opposition to the physicians of Baltimore. We must also credit a dentist, Dr. Morton, with the public use of ether as an anesthetic and with making it known to the world in 1846, thus taking the pain out of surgery.

We now appreciate that the diseases of the world are primarily or secondarily caused by microbic life as their normal function or through their abnormal situation, about 87 per cent of all deaths being caused by acute or chronic infection. The first life in the world was the microscopic chemical agents without which multicellular life would not have developed. They first dealt with the inorganic, later with organic and cellular structures. But a few of them, the proportion of natural criminals, can be classified as the cause of disease. Those causing diseases have increased and will continue to increase; as time passes, new germs will develop new diseases. We know now the bacteriologic mode of attack, and efficient health departments will quickly study the activities of the germs and keep them in control as has been done with the great destroying diseases of the past, such as the plague, cholera, smallpox and typhus.

The infectious diseases of childhood, including those of the teeth, have largely been removed. By efficient quarantine, scarlet fever, measles and diphtheria are, temporarily at least, prevented during the period of an epidemic, and blood tests show whether or not there is immunity to diphtheria, and if not, the blood resistance can be raised.

We thus readily see from the high death rate of children and adults in the past, why the average length of life was low, even though some did live out the century. In the sixteenth century, the span of life was only two decades; and even in 1850, it was only four. It took twenty-five years to raise the span to forty-five years, and now it is up to fifty-eight. Probably only a few years more will be added to the span of life and these slowly. They will be added by still greater control of contagious plagues and infectious diseases. The greater part of the next twelve years of man's life will be added by his individual efforts in looking after his own health, through better education in eugenics and hygiene and as to the conditions that cause impaired health, and the community will be interested in making life so much more worth while that its citizens may want to live.

There is a drawback to man's prevention of disease, since we save the fit and the unfit; while in animal life, the mental status is at least preserved by the loss of the unfit. The unfit human beings are preserved and they breed other unfit unless the condition necessitates confining them to institutions, such as reformatories, jails, asylums and prisons.

A few decades ago, before the many laboratory aids in diagnosis were developed, the medical men of the period had become almost uncanny in their ability to make correct diagnoses from detailed observations, their only resource. For the dentist who recognizes the broadness of his field, it might be well to call

to mind a few of the abnormalities that he may see if he is observant when he examines the oral cavity: manifestations of scarlet fever; measles; diphtheria; pernicious anemia; sprue; scurvy; rickets; congenital syphilis; lead poisoning; Vincent's angina and noma; actinomycosis; diabetes; uremic stomatitis; acromegaly; myxedema and cretinism; Addison's disease; lymphatic leukemia; pellagra; dermatitis herpetiformis, and purpura. Some of these may have a bearing on the task in hand and some may not, but they may be danger signals, indicating the necessity for a thorough examination by a physician. A suggestion from the dentist regarding the advisability of having such an examination may mean much, possibly life itself, to the patient.

Recently, a patient was sent in by a dentist for examination. The man, who was up and able to travel, had called on a physician, who advised him to have a few teeth out. His dentist advised him to have a general examination first. A very severe diabetes was revealed, and the pulling of teeth in this condition might have precipitated coma and death.

Talking of the interdependence of medicine and dentistry is like talking of the interdependence of medicine and surgery, or medicine and obstetrics. The practice of medicine includes dentistry and dentistry is the practice of a special branch of medicine, as is ophthalmology.

It may be going too far to say that all dentists should be doctors of medicine, but certainly all dentists should know much about the practice of medicine as a whole; and, conversely, all physicians should know more about dentistry, its importance and possibilities.

The modern dentist in practice concerns, and should concern, himself not only with the teeth but also with the jaws and the whole oral cavity. Local lesions in the oral cavity may be the cause, the effect or the sign of remote local, systemic or organic disease anywhere in the body, and some organic, systemic or local diseases may and do produce various signs in the oral cavity. Consequently, the dentist who looks into a patient's mouth will miss something of vital importance to his patient if he sees only teeth in their capacity of masticating and aching, as will the physician who examines his patient complaining of some local trouble, systemic condition or organic lesion if he forgets the possibility of an oral cause.

The mistakes of the oral specialist who forgets that his chosen field is a part of the whole body are as regrettable as those of specialists in other regional fields and as those of the general practitioner who forgets that certain systemic conditions arise from local disorders.

Dentists have developed their specialty wonderfully, making, in fact, several subspecialties out of their opportunity to study the mouth. We are reminded of the latest definition of a specialist as the man who knows more and more about less and less, and of the general practitioner as one who knows less and less about more and more. There is certainly more and more to be learned about everything for the beginner in the study of every subject, more now than twenty-five years ago, and there will be much more known in twenty-five years than there is now. It was forty years ago last spring that I graduated in medicine, and the members of my class thought that they had to study very hard. The student of today must continue to read and study in order to keep up with the

new things concerning his profession whatever it may be.

It is true that we have probably reached the near limit of muscular development and control. A man's brain, however, when compared with that of animals, is enormous, and it contains many areas concerning which we know very little. It is probable, then, that we are using only a small part of our brain today and that man can continue to develop through his brain for ages and ages. Intellectually, then, he is destined to be a wonderful man. It is to be feared that many persons use but little more brain than the animals, as they allow their glands and habits to control them.

We must all admit that the dental profession not only survived but also made marvelous progress alone. It is now necessary to study four years to learn about dentistry, and to continue to study, in order to make progress.

The cost of education today is very great, and the added burden of years of time is a hardship on the limited number of students who take up this profession. The American youth, unless he makes an early start, may lose his initiative. A physician's survey made of the students of Ann Arbor who graduated some years ago showed that the more successful had graduated and started their work by the age of 25 or even when two or three years younger. It is apparently a great waste of time and equipment to have long summer vacations. Assistants and associate teachers would be glad to carry on the educational work. There should be a week's rest between each two periods, but the studies in all lines of education should be in four quarters, and if certain students need an extra vacation, they should drop out for a quarter and not hold students back a year for the loss of three months. We should pay more attention in this country, as they do in Europe, to the fundamentals of medicine, in the first three years. The clinical and special branches in the fourth year should be taken either then or at a later period, but the diploma for graduation should be given after a three-year period. I believe that this is true of any of the specialties of medicine, and I look on dentistry as a specialty of medicine. We certainly cannot be fully familiar with any specialty until we know very thoroughly the fundamentals of the whole. If we carry our special training too far, uneducated cults slip in. Some think that it would be expedient for the ophthalmologist to break away from medicine and devote four years to the study of the eye with only a moderate application of the study of the whole as a preliminary. Every part of the body is dependent on the whole and to develop a specialty more fully is to study what constitutes health and disease. The ophthalmologist is very proud of his ability not only to diagnose diseases of the eye but also, with the ophthalmoscope, to see the pulsating arteries and veins uncovered by tissue and so to diagnose a number of general diseases of the brain, blood, kidney and heart.

During the last few years, we have come to appreciate how few yet how essential are the sixteen elements from which animal life is built, and as a great part of our food is from plant life, we note that animals and plants use the same group of material of the ninety-two elements which make up our universe. Variation from the normal blood volumes of these materials favors development of disease and gives foothold for germ life.

It is of the utmost importance to learn how to interpret roentgen-ray plates correctly as the local condition of teeth and jaws is but a local expression of the general health. In the individual diseases today, while they are infective in the end, the way is largely paved by what is taken as food and drink, lowering the resistance to some of the infections.

There is considerable truth in the old adage that man largely digs his grave with his teeth. A few years ago, there was a period of attributing rheumatism and many other diseases of nerves, joints, muscles and the heart to diseases of the teeth which might be eradicated. This is all very true, but without careful investigation, in many instances, good teeth were sacrificed without improvement in health.

Just now, focal infection is at a lower ebb than it was a few years ago. It was first started by Hippocrates, who described relief of rheumatism by extracting diseased teeth. Only a few people have made a real study of such troubles and, by research, secured the germ from the focus and reproduced the disease in animals, again recovering the germ and reproducing the disease. Well known physicians in this work have been Dr. Adami of Canada, Dr. Billings of Chicago, Dr. Judson Daland of Philadelphia, Dr. Rosenow of Rochester and Dr. Haden of Kansas City, and among the dentists there are Dr. Price of Cleveland and Dr. Hartzell of Minneapolis. Such research requires the greatest labor, care and effort. Many doubting Thomases have delayed the rapid spread or, at least the general appreciation, of this wonderful work.

All people need dentists to aid in the preservation of the teeth. Teeth must be cleaned, cavities filled and diseases of the gums cared for. Much more consideration is now given by dentists before they destroy nerves and crown teeth, and they appreciate the many diseases caused by focal infection from apical abscesses. Healed-in roots are to be found in 12 per cent of mouths in which full plates are worn. It is undoubtedly true that in diseases which recur in a similar manner from time to time, whether in the eye, nerves, joints or muscles, the seed is carried in our own kit bag of focal infection. The good dentist today has learned that there are other foci and advises further examination before the extraction of apparently healthy teeth.

The large tonsil in children may be a source of serious valvular heart disease. In adults, the large tonsil is large because of local resistance and only later, when smaller (size graded 2), with sealed crypts, becomes a source of distant joint infection. Diseases of the cornea and iris may come from a diseased tonsil or tooth, but in women probably many of them come from the uterine cervix; and in men, from the seminal vesicles and prostate. This should be borne in mind before a general extraction is made.

About 80 per cent of children should have some attention given their teeth. While I was on the school board, dental examination of the school children revealed that about 50 per cent of the children had teeth that needed filling, and 25 per cent needed extraction of one or more teeth. Children who grow up with such care will in later life try to secure similar or better care for their children. While we have many dentists, people in general do not avail themselves of the opportunity to care for their mouths, since only 20 per cent of the people of the world employ dentists. If we could but show the need for dentistry, we

would have work for more than double the present number of dentists.

Our country has wonderful dentists and many of them, as compared with the old world. Paris has one dentist to 6,000 or 8,000 persons, and the outer provinces have one dentist to from 20,000 to 38,000.

There is a growing interdependence between the dental and medical professions, and each in order to secure the best or at least good results necessarily becomes more or less dependent on the other.

Calcium in the world is largely handled by microbic life; that is, the laying down of limestone in the sea today is done by the most common organism of bacterial industry. It is thrown down as mud each year and becomes solidified gradually as time goes on, making the layers of limestone ultimately seen in the limestone quarry. This is also true of the development of stone in the kidney: it is built by microbes in the pelvis or in the calix. In experiments on dogs, under an anesthetic and with the most perfect preparation for asepsis, the bicuspid teeth were prepared by removal of nerve pulp and nerve root and the space filled with bacteria from the renal secretion of patients with stones in the kidneys, and the teeth were then crowned. The work was carried on by a dentist and a surgeon. In such experiments, roentgenograms of the jaws showed apical abscesses in from two and a half to five months, and roentgenograms of the kidney region showed the development of stones in the kidney in a period of from seven to ten months in 66 per cent. In 20 per cent more, the medulla of the kidney was filled with free calcium. These dogs had been tested and were perfectly healthy. The lesson was that stones in the kidney are caused by bacteria and that, after removal of stones from the kidney, patients must have extra care to relieve the mouth of all infection, of crowned teeth, of pyorrhea and of all sources of focal infection, to prevent the redevelopment of stones in the kidney. It was of interest to note that the stones in the animals caused by the microbes from renal secretions of patients with stones in the kidneys were just like the stones of the patients. Dark stones, light stones, phosphatic stones and coral-shaped stones were all reproduced by the type of worker, just as if some were cement workers, stone masons and bricklayers, and others were workers in terracotta and concrete. The great majority of persons die from acute or chronic infection, and checking the gross contagious diseases of the world permits a greater number to die an individual death from their own private infection. Today, we hear more of infections that are newer to us, such as sleeping sickness or encephalitis, really a contagious type of brain disease; and even disseminated sclerosis of the spinal cord, chronic nephritis, diseases of the ureter and bladder, recurring rheumatism of muscles and joints, chronic recurring diseases of the eye (iritis, retinal and choroidal trouble and corneal ulcer) and neuritis are some of the more common diseases which now we know to be due to infection. In fact, practically all of the cases in which disease recurs, each time acting like the disease when it first appeared or as it appeared later, emphasize that we are carrying the foci of disease within us, in a large number of cases in the mouth, usually in closed marginal pockets and the tonsillar crypts. We have the same type of germ as a rule as is found about the teeth in pyorrhea. Abscesses at the roots of pulpless teeth, and devitalized

and crowned teeth, all afford opportunity for infection leading to an early break in health, with the same germ found in secondary foci.

It is unfortunate that so few appreciate from what small causes diseases come. Unfortunately, sometimes their physician does not and neither does their dentist. Many persons are sick without realizing that they are sick because they are not suffering pain, and they are surprised, when they have an examination and an audit of the state of their health, to find that they have high blood pressure or trouble with the kidneys or heart. The roentgen-ray has been of enormous help in locating disease of the teeth and jaws. The vitality of teeth must be more carefully investigated. The roentgen-ray cannot furnish all the evidence that dentists or medical men think it does or should. The message left on roentgenographic plates can be read by comparatively few physicians who understand the reading of such plates. The dentist must accept the responsibility of all work done in the mouth, on the teeth, jaws and gums, in cases of infection. It must be remembered that the mouth is not the whole source of infective foci, and that after years of sickness, multiple foci may develop. In some cases, it may be necessary for the dentist to express his belief that more investigating should be done before fairly good teeth are sacrificed. I believe that as we all approach middle age and begin to lose our teeth one by one, we take a greater interest in the absolute necessity for their removal in the best interests of the preservation of health. More personal hygiene and improved heredity are the keynote to the health of the coming generation.

TREATMENT OF CLEFT LIP AND PALATE*

By FULTON RISDON, M.B., F.A.C.S., D.D.S., Toronto, Canada

DOUBTLESS, operations for cleft lip and palate were performed before 1776, but in that year Lemonier described it. Warren of Boston (1820), Sir William Ferguson, Van Langonbeck and many others influenced the treatment of this disease.

American surgeons, Truman W. Brophy in particular, have strongly influenced the treatment of this condition. This influence has been conservative, speaking generally, but recently certain surgeons in this country have shown more radical tendencies. On the European continent, the more radical operations are in favor; while in England, except for one school, the more conservative treatment has been popular with the more experienced.

The etiology is fairly well understood. The condition is due to the incomplete closure of the maxillary and nasofrontal processes *in utero* before the fifth week. We do not believe that syphilis has any bearing on the deformity, and certainly, with our present knowledge, no young mother should be worried by such a suspicion, as it has no scientific basis.

As to prenatal influence, one can only

*Read before the Section on Oral Surgery at the Midwinter Clinic of the Chicago Dental Society, Jan 24, 1928.

say that as the closure of the lip and palate occurs before the fifth week *in utero,* unpleasant sights could have no influence after that time, as the union of the clefts has already occurred. We then come back to heredity, and if the mother has or had a complete knowledge of her husband's and her own forebears for three or four generations, she would be aware of this condition in the family. Another fact to consider is that the public, as a rule, remember what they desire to and forget the unpleasant things, familial or otherwise. No doubt, insurance companies have suggested this convenient forgetfulness to the human mind, as they ask such questions in their applications, as "Is there cancer in your family, tuberculosis, etc.," and they might include cleft palate.

The general management of a case in our practice is as follows. If the child has a complete cleft of lip, ridge and palate, we advise that the cleft be closed before the third month and the lip at any time. If a cleft is in the lip only, the closure may be made at any time after the primary weight loss has been regained, on an average of about the twelfth day. We advise the parents that if any cases of measles, diphtheria, etc., are in the house or there is any likelihood of an exposure to such conditions, they should delay the operations for three weeks.

The child cannot nurse, so the breast pump should be used, and feeding should be supplemented by a suitable adulteration of cow's milk, usually starting with one portion of milk to two of water, with sugar and cream added. The feeding should be under the care of the family physician or pediatrician, and he will advise as to best time to operate, within certain limits. In all cases, the alveolar cleft should be closed before any operation is performed on the lip, or at the same time. Otherwise, the difficulty of such an operation to obtain bony closure is increased. The closure of the alveolar ridge can be accomplished in two ways: by pressing the two sections of the alveolar ridge together and holding them by a single wire; or by the other method, introduced by Dr. Brophy, by which double wires are passed above the teeth buds from malar eminence to malar eminence and another wire still further forward and in such a position that the wires may be twisted together over lead plates. Thus, pressure is made by taking up the slack in the wires, one wire of which is brought around to make pressure in front to hold the alveolar sections together. This works equally well for single or double alveolar clefts. The wires are removed in six weeks.

Dr. Brophy, that pioneer of this type of surgery, a man of whom we are justly proud and love to honor, first advocated this wiring operation. He has been criticized more at home than abroad, if possible, but we believe that, especially for the double alveolar clefts, it is the best treatment. Doubtless owing to my training under Dr. Brophy, I would favor this; but for some time I gave it up. Recently I have returned to my earlier ideas, with a slight change, in that, with a large bone pressure forceps, the lateral alveolar ridges are greenstick fractured toward the median line, the wires merely acting as a splint. In the double anterior cleft cases, the vomer has to be divided to allow the premaxillary bone to be replaced. With the greenstick fracture method, the premaxillary bone does not become so flat as with the earlier pressure method recommended by Dr. Brophy and, at the same time, this procedure saves considerable time and obviates the danger of the

breaking of the wires from overtwisting. I am fully aware that Dr. Brophy used this fracture method but recently had discontinued it.

The more common method of treating the alveolar cleft is to press the segments together and retain with catgut or silkworm gut passed through the bone with a heavy needle. The edges of the alveolar cleft are lightly freshened, but this is not really necessary. Perhaps more cases are treated by merely pressing the segments as closely together as possible and closing the lip, depending on the pressure of the new lip to hold the sections of the alveolar process together. Union of the segments is very important, and we should not be satisfied with mere approximation as, in later life, this fissure will spread if any orthodontic treatment is attempted, and most of the cases need it.

I am fully aware that this is a controversial point, but the ideal is bony or fibrous union of the parts. It is claimed that the shock is too great, that teeth are lost from pressure, that the soft and hard tissue sloughs from pressure and that the upper lip will be too flat. All of the foregoing objections are not real if the operation is properly done, but all these accidents have happened, owing largely to faulty technic. Recently, I reviewed a number of cases in which certain authors showed photographs illustrating the failure of such a procedure. One case still shows the premaxillary bone in the advanced position and another shows the upper lip too flat. Needless to say, these absolute failures in attempting this wiring method do not discredit it. I feel that these cases were unwisely chosen in an attempt to discredit an ideal method.

I do not wish to be misunderstood in that this double wiring is always necessary: frequently, in the small alveolar cleft, I use a single wire, but if the cleft is wide, I prefer the double wiring.

Many operations have been devised for the cleft lip, single or double, but they all may be divided into two types: (1) little or no vermilion border being removed, or (2) considerable of the border being removed. I find that this is a very difficult operation to obtain uniform results, and by that I mean, a well shaped nostril, not too long nor too tight, a lip and a good full vermilion border. As all these operations are fully described in the textbooks, I shall not attempt a detailed description.

The palate should be operated on toward the end of the third year or between the second and third year and seldom or ever before this time. If the operation is performed early, the tissues are friable and thin; whereas, by the second year, they are fairly thick. Furthermore, we seldom make a lateral incision in the primary cases, but generally find them necessary in the repairing of failures or secondary cases. It has been my habit to use lead plates and bronze wire for splinting the palate, perhaps taking off a little of the tension; and over a period of ten years, I have not had a failure, although occasionally there is a small hole at the junction of the soft and hard palate. The tension may be relieved by catgut or silk, but to date I have been splinting the palate as above stated.

In Europe, the very long lateral incisions are used in most of the clinics, but, in most cases, I cannot believe that it is good practice. Lane of London, uses flaps from the palate tissue. My objections to these methods are that the palate is too stiff and short. We raise the edge of the cleft with sharp elevators, handling the tissue as little as

possible, carrying the duller elevators well up to the tooth line and outward on the soft palate, and denuding the edge of the cleft. The bronze wires and lead plates are now introduced and the cleft closed with selected horsehair doubled and interrupted with the occasional mattress suture. The wires, or most of them, are tightened after all the sutures are in, and are left for eight days.

I believe that more failures occur in the palate because of rough handling of the tissue than from any other cause, but one should not forget to ascertain whether the child has been exposed to the infective fevers. If the operation is performed in a children's hospital, special wards and operating rooms should be provided, but the work is better done as far away from other children as possible.

The after-diet for this child is well left to the pediatrician, but, generally speaking, after a cleft lip operation, sterile water and 5 per cent glucose is given, 1 ounce per hour for one day, and feedings are started the next day, half strength, and rapidly increased. A laxative is given in the first feeding to clean the alimentary system of any swallowed blood.

The lip is first covered with petrolatum (vaseline), and the suture line is kept dry by cleaning with peroxid, if necessary, and drying with alcohol.

The baby that has undergone a cleft palate operation is treated quite differently. The last feeding is given four hours before operation, as in all cases, and while the patient is on the operating table, 500 c.c. of 5 per cent glucose is injected in the axillae. Orders are left in the ward that a laxative be given the first night to free the intestines of swallowed blood and that the child be placed on sterile water and 5 per cent glucose by mouth, one and a half ounces per hour for three days; whiskey drops, ten every three hours, and if restless, morphin sulphate, one two-hundred-fiftieth to one one-hundred-fiftieth grain, not to exceed five doses at three-hour intervals.

On completion of the palate and after eruption of the teeth, the orthodontist should be consulted, but, in my experience, it has been possible to do very little because of the financial condition of the parents. As the colleges do not care to assume responsibility for cases over the twelve months, college orthodontia, in our experience, has not been a success. It is to be hoped that, in the future, some arrangements can be made for free orthodontic services in the hospitals, but, for the present, we must leave the responsibility where it belongs, with the orthodontist, as he is fully aware of the need.

We do not favor obturators, except for the cases that have failed surgically.

SUMMARY

1. The child should be away from other children as much as possible for three weeks before operation.

2. If palates can be operated on without lateral incisions, a marked advantage accrues.

3. Union of the cleft of the alveolar process is the ideal result and should be attempted.

4. The dentist, and especially the orthodontist, must be consulted.

5. After operation, speech training will be necessary.

6. The best time to operate for closure of the cleft in alveolar ridge is before the third month; for the lip, any time after alveolar closure; for the palate, at about 24 months or later.

INTRAFOLLICULAR ENAMEL DEVELOPMENT*

By THEODORE B. VON BEUST, D.D.S., Louisville, Ky.

THE teeth used in this investigation were the molars of the pig. The subject of intrafollicular enamel development may well be approached with a few remarks on the origin and development of the molar follicles. Authors are not yet agreed on the genesis of these teeth, various possibilities being mentioned in the textbooks.

We will first study the origin of the third molar. Figure 1 shows the jaw of a young animal, the crypts of the second and third molars and the canal which contained the dental lamina (at *a*) being laid open. It will be seen from the size of its cavity that the third molar was in a very early stage of development, a small bud from the lamina having formed a crypt. The second molar, as compared to this one, has already grown to a considerable size.

Figure 2 shows these structures enlarged. A piece of cord has been inserted in the canal above and in the mandibular canal below. The upper canal we may for convenience call the gubernacular canal. It will be noted that the dental lamina continues posteriorly beyond the third molar through a canal in the bone; a fact which has not heretofore been shown to occur. It finally makes its exit from a foramen, which we will call the posterior gubernacular foramen. This opening is situated a few millimeters above the mandibular foramen in most jaws. It becomes obliterated after the eruption of the third molar. This posterior extension, after it leaves its foramen, joins the structures which enter the mandibular canal.

Figure 3 shows for comparison the lamina of an embryo calf with the second molar bud shown at *a*. The lamina in this case joins the mandibular structures inside the canal, the illustration showing its insertion in a segment of the mandibular canal content, which has been uncovered and withdrawn into view.

Figure 4 illustrates the further growth of the follicles of the pig. (Compare the size of the third molar crypt with the one shown in Figure 2.)[1] At *a* is the gubernacular canal between the two molars, and at *b*, its extension beyond

*From the Research Department of the School of Dentistry, University of Louisville.

*Working under grant from the Research Commission of the American Dental Association.

*Read before the Section on Histology, Physiology, Pathology, Bacteriology and Chemistry (Research) at the Sixty-Ninth Annual Session of the American Dental Association, Detroit, Mich., Oct. 26, 1927.

1. The follicles fill out their cavities completely. The bony septum between the follicles is often fenestered. Sometimes a bony septum is entirely lacking; for instance, the septum in one specimen was lacking between the cuspid and the third bicuspid germ, only a membranous partition being present.. The follicles are attached to their walls by very tender adhesions, excepting at the point of attachment of the lamina, where the parts adhere tenaciously.

the third molar. At *c*, the mandibular foramen is seen. On the significance of the posterior extension of the lamina beyond the third molar, I shall report later.

In Figure 5, we see the structures that were removed from the specimen shown in the foregoing illustration. At *a* is the mucus membrane of the mouth; at *b*, the posterior extension of the lamina. The follicles display no other connection with one another at any stage of their existence. They do connect, by means of their gubernaculum, with the soft tissues that cover the future site of the teeth at all times. This is not generally believed to be the case.

Figure 7 shows us an illustration of the tooth of this animal. It will be observed that it is made up of numerous cusps and subsidiary cusps. Roentgen-ray examination of its developing follicles was made. Figure 8 discloses the fact that calcification begins at segregated points removed from one another, the distance between the denticles corresponding approximately to the future position of the matured cusps. This observation disagrees in a measure with the teachings of recent textbooks,[2] which hold that the denticles are originally in apposition and subsequently travel apart. While a slight enlargement of the tooth crown may occur in this manner, it does not altogether explain the growth phenomena as observed in these specimens. The number of segregated denticles seen in this follicle may be computed at eight.

Fig. 1.—Mandible of a young pig, with bone removed to expose the crypts for the permanent teeth.

The first molar originates from the lamina in a manner similar to that of the second and third. Figure 6 shows the jaw of the pig at birth. The dental lamina connecting the posterior temporary molar with the posterior permanent molar is here shown at *a*.

FORMATION OF THE ENAMEL

Having viewed the exterior of the developing follicles, we will now proceed to consider their interior. For our purpose, we will study the third molar of the pig.

It will be seen that the anterior units entering into the construction of the crown are developed in advance of those situated posteriorly. The enamel of the anterior cusps will therefore be in a more advanced state of development than that

2. Noyes, F. B., and Thomas, N. G.: Textbook of Histology and Embryology, Philadelphia: Lea and Febiger, 1921, p. 114.

Fig. 2.—Enlarged view of Figure 1. *a*, posterior gubernacular foramen; *b*, mandibular foramen.

Fig. 3.—Mandible of embryo calf. *a*, bud for the second permanent molar; *b*, a piece of the marrow of the mandibular canal which has been withdrawn into view.

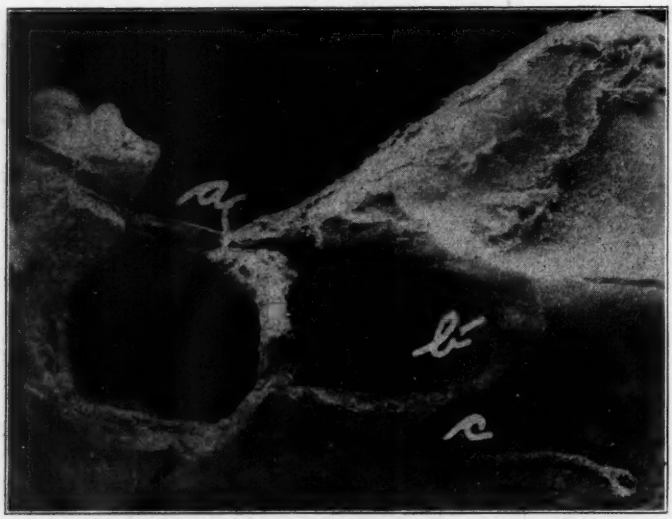


Fig. 4.—Mandible of pig, second and third molar crypts shown. *a*, canal for the dental lamina; *b*, posterior gubernacular canal with its foramen; *c*, mandibular foramen.

Fig. 5.—The tissues taken from the specimen shown in Figure 5. *a*, mucous membrane; *b*, posterior extension of the lamina; *c*, dental lamina.

of the posterior. This is to be borne in mind as we proceed.

Figure 9 shows a dried follicle which in age corresponds approximately to the one shown in Figure 8. According to the accounts of enamel calcification based on the work of Leon Williams and Hopewell-Smith, we would expect the cusps here seen to be covered with a few layers of formed enamel. While it is true that sections made with the microtome disclose the structural elements of enamel, an examination by physical means shows merely a mass of horny consistence which is only slightly impregnated with inorganic matter and which may be disrupted with the finger nail.

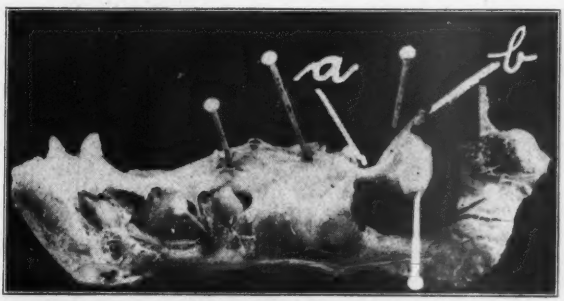


Fig. 6.—Pig at birth, with follicle of the first molar; *a*, dental lamina; *b*, posterior extension of the lamina on which the follicles of the second and third molars will be formed.

Figure 10 shows a follicle in a somewhat later stage of development. Its fellow of the opposite side was decalcified in formic acid, embedded in celloidin and vertically sectioned anteroposteriorly.

Fig. 7.—Third molar of the pig (enlarged).

Figure 11 illustrates one of these sections stained with magnesia-carmin. It will be seen that the enamel of the cusps of the more developed anterior region is

many times thicker than the dentin. No precautions were taken in any of these section specimens to support the enamel by an embedding process before decalcification. Preliminary embedding would have counteracted the shrinkage noted;

Fig. 8.—Roentgenogram of third molar of a young pig, showing segregation of the denticles.

but I am endeavoring to demonstrate that there is organic matter in sufficient amount to retain much of the enamel matrix. The projection in the upper left side of the picture is the sectioned posterior extension of the lamina.

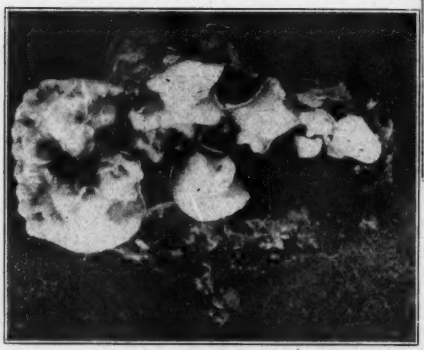


Fig. 9.—Dried follicle showing denticles (enlarged).

In Figure 12, we have an anterior cusp of the same follicle stained with hematoxylin. The space occupied by the enamel matrix almost equals in amount the enamel covering of erupted teeth.

I have long recognized the fact that enamel is not deposited in finished layers and I trust that I can make it evident that this tissue is deposited as a keratin-like substance containing only a minimum amount of inorganic deposit and that, at the stage here shown, it may be destroyed with the finger nail. The published accounts of its deposition are not supported by fact. Enamel is *not* laid down in calcified layers, the inorganic salts entering into the structure of this tissue being secondarily deposited probably by dialysis, in a horny matrix of great magnitude. The organic matrix deposited nearly equals in depth the enamel covering of the mature tooth be-

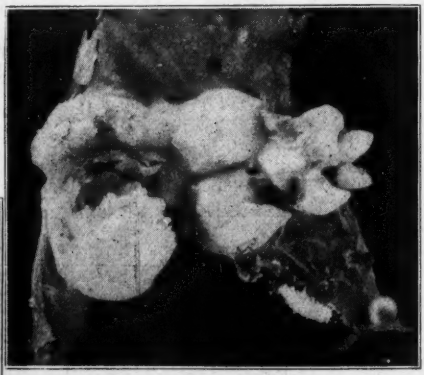


Fig. 10.—Dried follicle at a later stage of development.

fore calcification has progressed sufficiently to create a substance which a dentist would call enamel. There is, therefore, no solid enamel deposited—it becomes solid months after the formation of its matrix. That which has heretofore been taught about the "rhythmic" deposition of enamel, whereby the striae of Retzius are said to be formed, is based on a misconception of what actually occurs. How can it be affirmed that enamel is calcied "rhythmically" through about a half-hundred of its theoretical layers;

i. e., its lines of Retzius? I again wish to emphasize that enamel is calcified *en masse* and that the ameloblasts do not deposit finished layers of enamel.

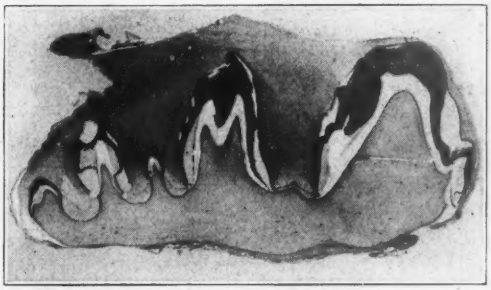


Fig. 11.—Section of a follicle at the stage of development shown in Figure 17. (Acid hematoxylin stain.)

In 1923,[3] I published suggestions for a division of enamel calcification. The intrafollicular stage was termed the protrophic stage. This may appropriately be divided into an early and a late protrophic stage. I illustrated my views at that time with ground sections made from the incisor of the ground-hog. Absorption of stain by all of the tissue elements of the enamel indicated a low degree of calcification.

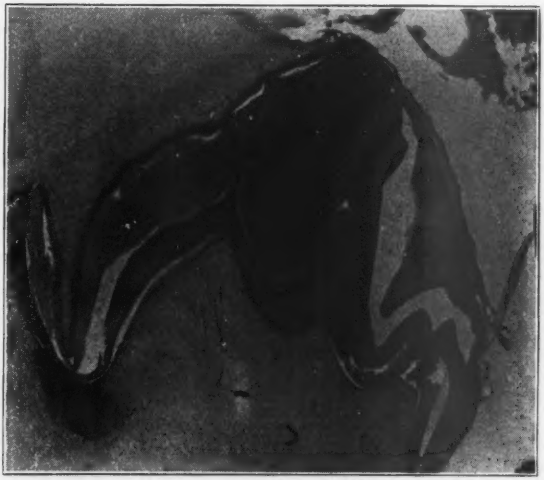


Fig. 12.—Enlarged view of an enamel cusp. (Hematoxylin stain.)

Figure 13 shows the mandible of a pig with the first permanent molar erupted.

3. Von Beust, T. B.: Physiologic Changes in the Structure of the Enamel After Tooth Eruption, J. A. D. A., 11: 396 (May) 1924.

The second molar crypt, immediately behind it, shows the position in the jaw of the follicle, which we will now study.

Fig. 13.—Mandible of pig, with second and third molar crypt exposed.

The follicle was fixed in an alkaline solution of formaldehyd (40 per cent), was stained for one month in magnesia carmin and was then subjected to the Weil process. The ground sections showed that the enamel was stained almost throughout, which indicated only a minimal degree of calcification. Figure 14, shows the effect of the staining, the enamel having become very nonactinic through the absorption of the red stain, appearing black in the illustration.

All teeth are "chalky," impregnable for stains and highly susceptible to decalcification by acids until their normal advent into the oral cavity occurs; whereupon, profound changes, characterized macroscopically by a sudden transition from the chalky to the translucent state, mark the beginning of the mesotrophic stage. A knowledge of the changes occurring at the time of eruption is important in the determination of the etiology of mottled enamel.

The behavior of young protrophic enamel toward acids is quite puzzling. It has long been known to microtomists working on this tissue that, under certain conditions, portions of the enamel would resist the acids ordinarily used for

Fig. 14.—Weil preparation of the second molar germ, taken from the specimen shown in Figure 13.

decalcification. Those opposing the view that enamel contains organic matter have contended that this resistent matrix is simply partially decalcified enamel. After much thought, the following experiment was tried. A third molar follicle of a pig, older than those previously referred to, was decalcified in formic acid.[4] Thinking that the omission of the washing process would insure a larger amount of enamel in case the latter conception were true, the specimen was embedded, without washing, immediately after decalcification.

Fig. 15.—Third molar follicle of pig; illustrating the enamel of a part of the talonid which escaped formic acid decalcification.

Figure 15 shows a section stained with picro-acid-fuchsin. The soft parts have

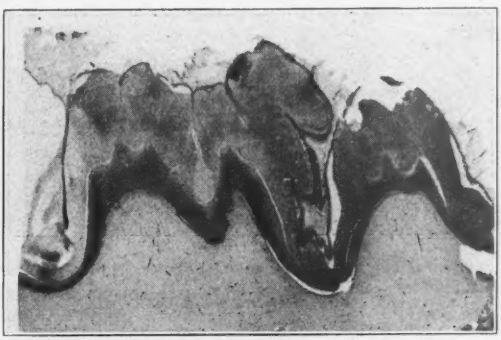


Fig. 16.—Section exposed to 5 per cent nitric acid for forty-eight hours, yet the enamel is still intact. (Acid hematoxylin stain.)

4. Decalcifying and fixing solution for follicles:

Water	500 c.c.
Formic Acid	50 c.c.
40% formaldehyd	50 c.c.

Three to five days immersion is sufficient for follicles in which the denticles have not yet coalesced. For older follicles, it will be necessary to renew the solution one or more times.

been defaced by calcific residue. The enamel of the more mature anterior moiety was removed by the acid, while that of a part of the talonid, being the less developed portion, remained nearly intact. The calcific deposits were removed from these sections by immersion in 5 per cent nitric acid for one hour. As the enamel matrix seen in the illustration withstood this treatment, the immersion in the acid solution was prolonged to forty-eight hours. The enamel matrix turned a brilliant yellow but stained well after a twenty-four hours' treatment with Buetschli's hematoxylin. The posterior cusps shown in Figure 16 were treated in this manner. In view of this behavior, it cannot be logically contended that we are here dealing with partially decalcified enamel.

Fig. 17.—Slowly decalcified enamel of man, showing distorted lamellae and tufts. At *a* and *b*, the enamel has separated from the dentin and is folded upon itself at *a*.

In 1911,[5] encouraged by the findings of Caush,[6] I injected and stained the enamel of freshly extracted teeth by capillary flow by way of the dentinal tubules. Since that time, I have affirmed in various publications that metabolic flow does occur in the enamel of erupted teeth. I moreover contended that the enamel rod substance of young teeth showed greater affinity for stains and was more susceptible to acids than was more mature enamel. Recent publications by Leon Williams[7] and Hopewell-Smith[8] therefore compel me to extend my remarks to cover posteruptive enamel. These authors do not consider many pertinent facts which have been disclosed during the past years. I call attention to the work of Mummery[9] and to my demonstration before this Section of the parallel rows of communication existing between the dentin and the enamel. As both of these writers continue to declare

5. Von Beust, T. B.: Arch. f. Zahnheilk., Nos. 1 and 2, 1912; Dent. Cosmos, 54: 659 (June) 1912.

6. Caush, Donald: Tr. 4th Internat. Cong., 1: 175, 1904.

7. Williams, J. L.: Dent. Cosmos, 69: 590 (June) 1927.

8. Hopewell-Smith, Arthur: Dent. Cosmos, 68: 639 (July), 360 (April), 468 (May) 1927.

9. Mummery, J. H.: Brit. Dent. J., 47: 473 (May 1) 1926.

that enamel is outside the pale of nutrition, I shall spend my remaining five minutes in reemphasizing that all metabolic changes in the enamel are not intrafollicular and that the title of my paper does not embody an inconsistency.

The fissurelike structures which C. F. Bödecker has named lamellae and the closely related figures from which the lamellae have been derived, which Caush[6] has named fan-shaped bundles of tubes, and C. F. Bödecker tufts, compose the parallel rows of capillary spaces described by me at length in 1926.[10] That these structures are not artefacts, as stated by Hopewell-Smith,[11] is proved by their behavior to slow decalcification processes, toward which they display greater resistence than the enamel rods. Into these interstices between the rods, there appears to be diffused a keratin-like substance which subsequently calcifies. Figure 17 shows one of the effects of a slow decalcification of a ground section of an adult human tooth.[12] The enamel matrix at the stage of decalcification seen in the photograph has become transparent and flexible, quivering in response to the vibrations of the building. In the applying of the cover-glass, the enamel has become partially displaced and torn from its attachment to the dentin. The tufts and the lamellae, otherwise intact, are distorted and deflected from their normal course; which clearly indicates that the enamel has assumed the consistency of a flexible membrane. By prolonging the exposure to acid, the rod matrix-substance, the tufts, the lamellae and Nasmyth's membrane, with which the lamellae are usually continuous, are the last, in the order named, to be affected. Nasmyth's membrane, as is well known, will resist far greater acid concentrations than are here being used. In 1905,[13] C. F. Bödecker published his acid-celloidin embedding method by means of which fixed preparations of the lamellae can be produced.

As indicated by their behavior to acids, the parallel rows of capillary spaces between the rods represent filled interstices, through or around which stains may be caused to flow into the enamel. They are organic in nature and are not artefacts produced by the hydrochloric acid radical of the monohydrochlorid of rosanilin, as contended by Hopewell-Smith.[14]

129 East Broadway.

10. Von Beust, T. B.: Capillaries of the Enamel and Their Relation to Mottled Teeth, J. A. D. A., 13:980 (July) 1926.

11. Footnote 8, first reference, p. 657.

12. Slow decalcifying mixture for enamel cross-sections:

Water	400 c.c.
40% formaldehyd	30 c.c.
Formic acid	3 c.c.
Lactic acid	2 c.c.

Attach sections of about 30 microns to a slide with one or more drops of warmed 50 per cent solution of gelatin in water. Allow to set for about ten minutes. Place specimen in a Petri dish and cover with the fluid. Decalcification is complete in from one to three days, depending on the thickness of the specimen. For photographic purposes, a drop of the gelatin may be carefully added and a cover-glass applied when the suitable degree of decalcification has been reached. To make permanent preparations, invest on a slide in a drop of celloidin of about 10 per cent; remove air bubbles, if necessary, by exposing the preparation to ether under a bell jar. Pass down through graded alcohols to the decalcifying solution. After the decalcification, pass up through graded alcohols, absolute alcohol a few seconds, creosote plus xylol until cleared, and invest in balsam. During these manipulations, the celloidin may part from the slide, in which event the specimen is handled as a celloidin section.

13. Bödecker, C. F.: Ztschr. f. Wissenschaftl. Mikroskop., 22: 190-192, 1905.

14. Footnote 8, second reference, pp. 375-376.

VINCENT'S INFECTION FROM THE PATHOLOGIC AND BACTERIOLOGIC POINT OF VIEW*

By T. A. HARDGROVE, D.D.S., Fond du Lac, Wis.

WHEN dealing with Vincent's infection, we are not dealing with a new disease. It has always existed, but, as with many other diseases, when people are congregated in great numbers, the organisms increase not only in number but also in virulence. For many years, there has passed through our hands Vincent's infection that was unrecognized, misdiagnosed and mistreated, being regarded as symptomatic of syphilis, tuberculosis, scurvy and other diseases.

While Vincent's infection is a local disease, we must not regard it as entirely incapable of producing a systemic disturbance. For instance, a patient may have an elevation of temperature, feel sick, complain of pain in the back and even show albumin in the urine. This condition can be brought about as a result of a mixed infection often associated with Vincent's. The resulting lowered vitality and possible disturbance of protein metabolism could cause the nephritis. The majority of such patients will be relieved as soon as the Vincent's infection subsides. But such a patient may go to a physician for treatment of a kidney disturbance alone, and the cause of his symptoms may not be recognized.

The patient who complains of bleeding gums, and thinks he has pyorrhea, is liable, in these times, to have a case of Vincent's infection. In the incipient cases, the oral examination will reveal a slight ulceration, usually beginning in the region of the third molar or any place about the teeth, with a grayish yellow surface not easily removed, but bleeding easily when disturbed. There is a distinct odor unlike that of other infectious or ulcerating diseases, and especially unlike the odor of pyorrhea. Once detected, the odor alone is almost sufficient to establish the diagnosis.

When associated with the teeth and gums, Vincent's infection operates beneath the free margins of the gums. The organisms are acually beneath the margins and beneath the ulcerating surface. But it is not entirely confined to the gums, as it can develop in any hidden or covered locality, such as the folds of mucous membrane back of the third molars and the crypts of the tonsil. It can even develop on the free mucous surfaces of the gums and cheeks. The more virulent the organism, the more likely it is to operate on a free surface.

There is no necessity for confusing the diagnosis of Vincent's infection and pyorrhea in this day when we can so easily and quickly identify the Vincent's organism by the use of dark-field microscopy, placing some of the material from the infection on a slide with a drop of physiologic sodium chlorid solution and examining it with the dark-field attachment. The number of spirochetes present should be noted, and their motile activity, as these two factors determine

*Read before the Odontographic Society of Chicago, April 5, 1928.

to some extent the virulence of the organism. If we make an examination after instituting treatment for a few days, we notice that the organisms are diminished in number and of much slower motility. In the material taken from pyorrhea infections, we may find some Vincent's organisms, but in fewer numbers and of less activity. While a patient may have both conditions at one time, I do not believe the presence of pyorrhea makes it any harder to cure Vincent's infection.

Because it may attack the tissues of the throat, Vincent's infection is often mistaken for diphtheria. Undoubtedly, many of the cases diagnosed as diphtheria in the past were nothing more than Vincent's infection. The membrane of Vincent's infection on the tonsil is not so white as that produced by the Klebs-Loeffler bacillus and is more easily removed.

Vincent's infection invades the respiratory tract, involving the lungs, where it may be mistaken for a tuberculous process. This is not likely to happen, as the organism is to a very great extent anaerobic, and does not thrive where there is a free circulation of oxygen.

The beginner may sometimes mistake a mercurial stomatitis for Vincent's infection. Here, the history will clear up the diagnosis. But it must be remembered that a patient may have Vincent's infection and either syphilitic sore mouth or mercurial stomatitis at the same time; and that the organisms of Vincent's infection and syphilis are both spirochetes, and somewhat alike in appearance.

The causative organism of Vincent's infection is the spirochete of Vincent, similar to *Spirochaeta pallida.* (I recognize its symbiosis with the fusiform bacillus, but for diagnostic purposes the recognition of the spirochete is sufficient.) These two spirochetae, when seen under the microscope, resemble each other so closely that one would wonder why they do not cause the same symptoms. This resemblance illustrates the wonderful accuracy of biologic differentiation. While they may seem very much alike in appearance, the two organisms produce lesions, symptoms and diseases that are not at all alike.

Spirochaeta pallida, because it is to some extent an anaerobic organism, produces a chancre in its effort to keep out of the blood stream. The incubation of syphilis in the body requires from thirty to sixty days, while that of Vincent's infection can be almost spontaneous, occurring at the moment of contamination. There are those who believe that it takes a longer period, even twenty-one days, for the incubation of Vincent's infection; but, in reality, the organisms immediately infect the external surface and do not have to be in the mouth of the patient for any great time before signs of their presence are manifest. The idea that these two diseases are related is a mistaken one. While the organisms may appear to be alike, the end results of their activity are entirely different.

As the organisms are on the surface, it is easy to isolate and observe *Spirochaeta vincenti* with the microscope. But even if we know that we are dealing with a syphilitic lesion, it is difficult to secure a specimen of *Spirochaeta pallida,* because it burrows into the tissues and remains there.

The Vincent's organism has a wavelike corkscrew appearance. *Spirochaeta pallida* has almost the same appearance but has more acute angles. If we locate *Spirochaeta vincenti* in a dark field, we observe bacteria of rapid motility being formed and dying under our eyes. If we are searching for *Spirochaeta pallida*

and accidentally find it, we see organisms of the acute-angled type and of much slower motility.

Vincent's infection is contagious, and clean mouths seem to be not much more immune than mouths poorly cared for. Undoubtedly the greatest sources of infection are the common drinking place and eating utensils in restaurants, and those involved in personal habits. It is not common among children, for the reason, I believe, that their tissues are richly supplied with oxygen by the rapidity of the blood flow. It reaches its greatest activity in the adult, but does not seem to affect old people particularly, perhaps because they are less active and do not come in contact with so many sources of infection.

The disease is a local one and does not cause a septicemia. The spirochete of Vincent is never found in the blood stream, for the reason that all anaerobic organisms more or less avoid the blood stream because of its oxygen-carrying power. We do not usually find the organisms in infected antrums nor in the nasal or postnasal regions. Neither do they appear in the urine. We do not find them in animal life, and it seems almost impossible to inoculate the mouths of rabbits and guinea-pigs.

The manifestations are local, and, as in most other diseases, the organisms have their particular tissue of choice. Their growth is materially enhanced in a location where the free circulation of air is to some extent restricted. An external surface where oxygen is not present in large quantities is the location of their choice. So the hidden places of the mouth furnish optimal conditions for the spirochete of Vincent. The duration of the disease is entirely dependent on the resistance of the patient and the effort that is made to cure the condition.

While syphilis is hard to cure, Vincent's infection is absolutely curable. Arsphenamin or neo-arsphenamin will eliminate the organisms by the active germicidal effect of the arsenic. We can isolate the infected area and apply either directly into the pocket. But the arsphenamin treatment is an expensive, inconvenient and indefinite method of cure.

The obvious method takes advantage of the fact that the organism does not thrive in the presence of an excess of oxygen. This brings us to a consideration of the best method of supplying oxygen to the diseased tissues. Many incipient cases are cured simply by the frequent application of hydrogen peroxid. Sodium perborate is supposed to act as a specific by forming hydrogen peroxid when in solution, but it is not active enough to be really considered a specific or to bring about a sudden termination of the disease.

I have worked out the following procedure, which I know will cure the disease. If we apply directly to the point of infection a certain amount of chromium trioxid by carrying it on the point of an instrument that will go into the pocket, and then have the patient hold a mouthful of hydrogen peroxid, there will be a copious evolution of nascent oxygen, with almost spontaneous relief from the infection and practically instant relief from pain. The patient will expectorate a blue-black fluid which is formed as a result of the reaction between the chromium trioxid and the hydrogen peroxid.

The reaction is explained in this way: Chromium trioxid and hydrogen peroxid are both good oxidizing agents. Chromium compounds can exist in various stages of oxidation with corresponding changes of valence. The higher the valence of the chromium, the more oxygen it can unite with. These higher-oxygen compounds are very unstable, and

tend to decompose readily, liberating their surplus oxygen and being themselves reduced to compounds poorer in oxygen. The chromium trioxid combines with the water in the saliva to form chromic acid (orange color). The hydrogen peroxid oxidizes the chromic acid to perchromic acid (dark blue). Then, the perchromic acid and the hydrogen peroxid mutually decompose, with the evolution of nascent oxygen from both of them. By this decomposition, the perchromic acid is reduced to chromic oxid, but the chromic oxid combines immediately with some of the hydrogen peroxid to form the brown solution which remains when the reaction is completed outside the mouth. When this reaction is carried out *in vitro,* a temporary green color may be noticed, due to the presence of a dilute chromic sulphate solution formed as a result of the reaction between the chromium and the small amount of free sulphuric acid present in the hydrogen peroxid solution.

According to the chemical equations, the weight of the oxygen given off in this reaction is equal to almost one-third the combined weight of the chromium trioxid and pure hydrogen peroxid used. To liberate that amount of oxygen would require a much greater weight of sodium perborate, and even then the evolution of oxygen would be much slower because sodium perborate is only slightly soluble. At mouth temperature, chromium trioxid is about forty times as soluble as sodium perborate, which accounts for the rapid reaction of the former with hydrogen peroxid.

The following equations express the changes which take place. The final equation can be found in Dammer's Handbook of Inorganic Chemistry, an authoritative German text, in volume 3, page 534.

1. Chromium trioxid ($2CrO_3$) + water ($2H_2O$) yields chromic acid ($2H_2CrO_4$).*

2. Chromic acid ($2H_2CrO_4$)* + hydrogen peroxid (H_2O_2) yields water ($2H_2O$)* + perchromic acid ($2HCrO_4$).*

3. Perchromic acid ($2HCrO_4$)* + hydrogen peroxid ($2H_2O_2$) yields water ($3H_2O$) + chromic oxid (Cr_2O_3) + 6 atoms of nascent oxygen (6[O]).

4. Chromium trioxid ($2CrO_3$) + hydrogen peroxid ($3H_2O_2$) yields water ($3H_2O$) + chromic oxid (Cr_2O_3) + 6 atoms of nascent oxygen (6[O]).

The important point is that by using this combination of a highly soluble oxidizing agent with hydrogen peroxid, a large amount of nascent oxygen in its aggressive atomic state is instantly liberated at the very point where the anaerobes are concentrated, and is apt to destroy all or nearly all the bacteria in the field of infection by uniting with their constituent elements and by-products to form inert compounds. The other products of the reaction do not seem to harm the tissues nor retard the progress of the case in any way. They may, in fact, be of positive assistance. A purplish black deposit is left on the teeth and about the lesions on the mucous tissues. This disappears in from three to four hours, the length of time depending on the amount of chromic acid used. This deposit must be the result of a reaction with some substance present in the mouth, as it is not produced when the reaction takes place outside the mouth. At present, I am not prepared to say what the exact nature of this deposit is. Besides leaving oxidation products and precipitating the soluble albumins and chlorid of the secretions, chromic

*Substances which are formed and are entirely used up during the reaction.

acid and perchromic acid would be capable of entering into very complicated reactions with the human tissues.

This treatment is convenient and specific because it meets in several respects the peculiar conditions of the Vincent's infection. The organism is anaerobic, and this combination releases sufficient oxygen to destroy it. An instantly effective treatment is desirable, for obviously no medication can remain for any length of time in the mouth, nor should any medication be applied continuously. The chromium trioxid can be carried easily to the deepest recesses of the infected tissues, is instantly and highly soluble and reacts rapidly and vigorously with the hydrogen peroxid, so that the nascent oxygen at the moment of its liberation is in direct contact with all parts of the area of infection and can combine directly with the bacterial substances. Of course, the healing process necessary where any tissue has been attacked follows the treatment. But one or two treatments, three at the most, at intervals of twenty-four hours, seem to destroy all micro-organisms of the Vincent type.

DISCUSSION

P. G. Puterbaugh, Chicago, Ill.: My experience with Vincent's infection has in certain instances been more disappointing than that of Dr. Hardgrove. In diabetic patients, this infection has a great tendency to develop into noma, with a fatal termination. In one case that came to the college clinic a few years ago, in which smears showed typical organisms, the patient, a man, aged 72, had a waxy, yellow skin and a rapid pulse. His only complaint was of pain and soreness in his teeth and gums; however, he died two days later of septicemia. Vincent's infection should be divided into two forms: the acute form, which Dr. Hardgrove has classically outlined, and the chronic form, which presents an entirely different picture. Chronic Vincent's infection is characterized by a low grade hypertrophic gingivitis and destruction of the interdental papillae. The patients complain of a feeling of uneasiness and mouth discomfort, with a wedging sensation in the interproximal spaces. In such cases, it is extremely difficult to eradicate the organisms because of the deep gingival crevices, which result from the hypertrophied condition of the gum tissue. I have been most successful in the use of oxidizing agents in the treatment of all forms of this infection. Chromic acid and the various anilin dyes seem quite satisfactory in some cases, but in others they have been disappointing. The combined staining and oxidizing effect of the remedy suggested by Dr. Hardgrove should make it a valuable addition to our armamentarium. We should remember that Vincent's organisms are often present in gingival crevices when there is no visible ulceration, and that persistent bleeding of the gums is frequently from this cause.

Edgar D. Coolidge, Chicago, Ill.: One point I thought was not made very clear: that the two organisms are active together and that the disease is not recognized unless you have the two organisms, the fusiform bacillus and the spirillum. Just how prevalent the infection is has not yet been established. Surprising statements are appearing in the literature at present that we find these organisms in a large percentage of normal mouths, but when they appear together we are apt to have the condition that has been described by Dr. Hardgrove. In just what percentage of what we have considered our typical pyorrhea cases the organism may be found has not been given enough consideration. We find, if we follow the literature, that a large percentage of pyorrhea cases will show Vincent's organisms, the two kinds, in places, and in almost all cases, one or the other, the spirillum or the fusiform bacillus. They are both anaerobic; the spirillum is motile, the fusiform bacillus is not. So it is quite possible that it requires the two of them to make a lesion, one to attack the tissue and the other to act somewhat in combination with it, they together producing the flat ulcer. I have seen recently an article reporting the examination of a series of mouths, in which as high as 80 per cent showed these organisms present, without any definite lesions. I have found in my own cases that I did not need to take smears from the ulcers that showed infection to get a

positive picture of the organism. The ulcers, of course, are easily recognized. The patients generally come to us with the complaint of bleeding. There seems to be a considerable difference in the treatment of these cases. Some respond rather quickly, just as suggested, with a few treatments. These are more apt to be cases that are localized, but not the chronic type, as Dr. Puterbaugh has mentioned. Those chronic cases in which the organisms can be recovered from almost any area in the mouth are exceedingly difficult to handle. If we dismiss the patient too soon, in a short time there is a recurrence, discouraging to the patient and distracting to the operator. I approve of the treatment recommended. I have used it, in a little different way. I never have applied the chromic oxid directly to the area. I have always had it made into a 7 per cent solution, but that is as strong as I have ever applied it. Ten per cent is as strong a solution as I have ever seen recommended. I presume that it is applied in the crystal form. The solution is applied directly to the tissues. The treatment that I have used with the exception of the chromic acid treatment, was suggested by Dr. Black in his text and in his writings of some years back, in regard to ulcerous stomatitis. When he first discovered that the bacteria were anaerobic, he immediately applied the oxygen treatment in the form of hydrogen dioxid. The treatment consists in giving the patient small doses of calomel, a quarter grain after each meal and a quarter grain before going to bed, that is, 1 grain a day, taken three days in succession, followed each morning with a saline cathartic, three days in succession. The local treatment prescribed for the patient is 3 per cent commercial hydrogen dioxid, with instructons to use every four hours, washing freely for several minutes, keeping it in contact with all tissues of the mouth; and repeating this, not for three days, but continuously for a period of two or three weeks, even after all local evidences of the disease has passed. In addition to that, he recommended applying a protective compound tincture of benzoin over these areas after washing with the hydrogen dioxid. This relieves the soreness and protects the affected area from friction of the tongue and food for a few hours. By washing with the hydrogen dioxid, the tissue is impregnated with oxygen as much as possible. Protecting it with compound tincture of benzoin relieves the condition and gives a little more satisfactory result. During the war, chromic acid was recommended, and it was suggested to me by my associate, who returned from the war. I tried it and found it speeded up the result. I have been using the 7 per cent solution of chromic acid. Chromic oxid dissolved in water makes chromic acid. Using a 7 per cent solution, I paint the area rather generously. Suspected areas where the bacteria can easily lodge, I coat over with the compound tincture of benzoin, then prescribe the other treatment suggested, by all means urging the frequent and constant use of hydrogen dioxid diluted with equal parts of water. The majority of cases will respond quickly and without much difficulty. If the case shows considerable pyorrhea, and many pockets in the mouth, it is very difficult to get the mouth free from the bacteria. At just what point it is advisable to dismiss the patient is something I have not yet been able to determine. The time of incubation is suggested as three weeks. I do not know just how long incubation requires, but we need more information, and we certainly should carry our treatment over the period of incubation so there would be no recurrence from a reinfection. As it would be wiser to treat the condition a little too long than to stop treatment too soon, I instruct the patient to continue to use the hydrogen dioxid, at least for a month. I would mention three oxidizing agents that are useful. The chromic acid yields the largest percentage of oxygen—about 24 per cent of oxygen in chromic acid. In potassium permanganate, there is about 16 per cent available oxygen. Potassium permanganate has been recommended as a wash. I never have used it for fear of discoloration, but it is claimed that it will not discolor permanently. The third is sodium perborate. That yields about 2 per cent of oxygen. It has one thing to recommend it: It is a powder that can be easily applied and does not injure the tissue. It can be made into a thick paste with water and rubbed into the spaces between the teeth and left there for a time and afterwards washed out. It does not produce an amount of oxygen strong enough or rapid enough in action to destroy the tissue, while the chromic acid is a little severe and does melt down the surface of the tissue a little, and we get a little more exposed surface between the teeth than there was originally.

I noticed in the article[1] a few weeks ago that sodium perboiate has been recommended by some of the European men who have had considerable experience with it. Dr. Hardgrove spoke of arsphenamin and neo-arsphenamin. I have never used it by injection, but have applied it locally, and I find it is a very useful remedy. A 5 per cent solution of neo-arsphenamin in glycerin was used. It has a tendency to reduce the number of spirilla. The fusiform bacillus responds very quickly to the oxygen treatment. The spirilla do not respond so quickly, in my experience, but I find that neo-arsphenamin will reduce the number of spirilla more rapidly in those obstinate cases. I do not believe it is possible to eliminate the spirilla entirely because they are more or less common in the mouth. If we reduce the number of spirilla to a more nearly normal count and eliminate the fusiform bacilli I believe that we have eliminated the danger and corrected the disease.

Don M. Gallie, Jr., Chicago, Ill.: The thing that we are most interested in is the treatment of Vincent's infection. I have the history of practically everything known to the pharmacopeia that has been used in this condition. I think that we get just about the same results from all of them. The disease in a very acute stage is easy to clear up, but the problem, as has been so aptly stated, is the recurrence of the disease. I have found no agent, mechanical, therapeutic or medicinal, that has been able to eliminate this disease when it recurs. As to the use of arsenic intravenously, as spoken of by Dr. Coolidge, I have knowledge of two or three persons who are undergoing very thorough antisyphilitic treatment and at the same time, suffering from Vincent's infection. The acute symptoms of the disease were easily controlled by intravenous injections of arsenic, and yet as soon as these were continued, the cases showed a tendency to recurrence. I have never used the method of treatment Dr. Hardgrove describes and will be glad to try it. The problem, I have found, is not in curing the acute symptoms of the disease so much as it is in dealing with the chronic and recurrent potentialities of the disease.

Dr. Hardgrove (closing): I am not advocating peroxid as a treatment. I am laying down the principle that nascent oxygen will destroy the Vincent's organism. I am not paying so much attention to the fusiform bacillus as I did because we do not always find it present, but we have Vincent's infection just the same. Dr. Puterbaugh has called attention to the fact that probably a lot of the cases of gingivitis might be due in the beginning to, or maybe their chronicity is brought about by, the presence of Vincent's organism. I sometimes wonder if in the beginning pyorrhea was not brought about by Vincent's organism. It looks reasonable, but I do not wish to make a positive statement to that effect. I do not think anybody has been able to isolate the Vincent's organism from the blood stream. In those patients that died, probably the blood culture might show the regular streptococcus infection. I am inclined to think that it would. One objection to the continuous use of hydrogen peroxid is that it irritates the tissues and the continuous irritation of the epithelial tissue is not a good thing. If we prolong the use of hydrogen peroxid, we may stimulate an activity that we do not want. Dr. Coolidge referred to chromic acid cauterizing the tissue. Of course, we wish to dissolve the ulcerated surface, but that does not occur to the extent that he seems to think. We immediately get away from the cauterizing effect by the reaction. The moment we use the hydrogen peroxid, a new substance is formed. We have the chemical reaction in the mouth instead of outside. The process I have spoken of can be used two, three or four times and a culture can be made, and we will not find any organism and the patient will improve. We may examine again a month from that time, and there will be no active Vincent's infection, but we can find some of the organisms. We may find the fusiform bacillus, too. We make a dark-field examination for Vincent's, as a rule, and stain for the fusiform bacillus. I have found them many times in the same mouth, with no active disease. We talk of curing tuberculosis. There is no cure. We arrest the process, and the patient lives. The general principle that is involved in the treatment of every other bacterial disease is involved here. Immunity of the patient enters into consideration. Why is it that a patient for a time has no trouble, and suddenly the disease flares up? It is because the patient's immunity to that particular disease, even though it is local, is reduced, and when it is reduced, the virulence of the organism is increased. It is the

1. Loeb, Virgil: Trench Mouth, Bull. Chicago Dent. Soc., 9:7 (Sept. 2) 1927.

virulence that produces destructiveness in all diseases. When the symptoms have all disappeared and the patient is well, we have reason to believe that he is cured. I have seen healthy mouths, well cared for, but containing the organisms which are not doing any harm because they are attenuated. Prophylaxis is an important thing, but I do not think that with Vincent's infection it should be instituted at once. Attack the bacteria first, then give your attention to prophylaxis. When we try this, if we follow out the technic, you find the result as I have said it would be. I have seen some cases of Vincent's infection of six months' duration, and still active. Where the acuteness leaves off and chronicity begins is a nice demarcation. And of course, as Dr. Gallie said, when a patient has a mixed infection, such as syphilis, and we treat this infection, the patient's natural immunity will rise to a better level. When syphilis is untreated for a time, the patient's normal resistance is reduced. We have to take into consideration all these associated things. There is a great tendency to forget the bacterial origin of disease; nevertheless, bacteria have always been here and I believe they are going to stay for a long time. It is a question of how we are going to handle them.

DENTAL CARE AND THE FAMILY BUDGET*

By MICHAEL M. DAVIS, Ph.D., New York City

DOES the average man get the dental service he needs? How much does he pay for it and how much can he afford to pay for it? It has been estimated that, under ordinary conditions, one dentist can take good care of the teeth of not more than 500 persons, or 120 families. In the whole United States, there not more than 60,000 dentists, or one to every 2,000 persons. Even in the larger cities, the ratio of dentists is less than one to 1,000 persons. It is a matter of common knowledge that a large portion of the population do not get sufficient dental care. How much of this is due to lack of intelligence? How much to lack of dentists? How much to lack of money?

These questions are of great practical interest to dentists as well as to the lay public, and the purpose of this paper is to present some facts and suggestions regarding the economic side of dentistry from the standpoint of the consumer.

Probably few persons in these prosperous times appreciate how unevenly incomes are distributed in the United States. From the authoritative studies of the National Bureau of Economic Research, we find that of 37,569,060 individuals, in 1918, about a third were estimated to have annual incomes of from $1,000 to $1,500. The figures showed that 86 per cent of all persons in the United States had incomes of less than $2,000, 94 per cent less than $3,000, 98 per cent less than $5,000 and only 0.66⅔ per cent over $10,000. Later estimates indicate that the distribution of incomes has not substantially changed since 1918.

Thus, the great majority of self-supporting American families are living on incomes of a little above or a little below $2,000 a year. The State Housing Commission of New York estimated recently that two-thirds of the families in New York City had incomes of less than $2,500. Display and extravagance attract attention, but it is not upon the basis of those who drive Cadillacs and

*The assistance of Miss Margaret Lovell Plumley in the preparation of this article is gratefully acknowledged.

TABLE 1.—ANALYSIS BY THE UNITED STATES BUREAU OF LABOR STATISTICS

Income Group	Number of Families	Average Per Cent in Families	Average Yearly Expenses per Family for: Food Amt.	Food Per Cent	Clothing Amt.	Clothing Per Cent	Rent Amt.	Rent Per Cent	Fuel and Light Amt.	Fuel and Light Per Cent	Furniture Amt.	Furniture Per Cent	Miscellany Amt.	Miscellany Per Cent
Under $900	332	4	$372	44	$112	13	$122	15	$ 57	7	$ 30	4	$150	18
$900 and under $1,200	2,423	5	456	42	156	15	150	14	64	6	48	4	201	19
$1,200 and under $1,500	3,959	5	516	40	207	16	180	14	73	6	62	5	262	20
$1,500 and under $1,800	2,730	5	572	37	257	17	207	14	79	5	84	6	335	22
$1,800 and under $2,100	1,594	5	627	36	307	18	232	13	87	5	97	6	404	23
$2,100 and under $2,500	705	6	712	35	384	19	248	12	93	5	117	6	500	24
$2,500 and over	353	6	860	35	503	20	260	11	102	4	133	5	608	25
All incomes	$12,096	5	$549	38	$248	17	$187	13	$ 74	5	$ 73	5	$306	21

fill orchestra chairs that family budgets can be estimated for dental service, or for anything else.

The Prince of Wales is said to have been charged $2,000 for two fillings. A prince's tooth may be worth $1,000. But how does Mr. Average Man figure his dental expenditures?

The United States Bureau of Labor Statistics made in 1918-1919 a significant analysis of the expenditures of 12,096 families selected from all types of wage earners in all sections of the country, with incomes ranging from $900 to $2,500 and up, with less than 400 in the top group whose incomes averaged as much as $2,790. These families each consisted of about five persons. Table 1 shows the division of their expenditures.

TABLE 2.—EXPENDITURES FOR DENTAL CARE AMONG 12,096 FAMILIES

Income Group	Av. Expenditure per Family for Dental Care
$ 900	$ 1.99
900-$1,200	4.59
1,200- 1,500	6.93
1,500- 1,800	9.63
1,800- 2,100	11.31
2,100- 2,500	13.81
2,500	17.84
All incomes	8.23

According to this table, the average family allotted about 20 per cent of its income to the "miscellaneous" division, or from $150 to $600, according to the size of the income. What do these "miscellaneous" items include? Almost everything it seems that goes to make up living—amusements and vacations; education, newspapers, magazines, books, school tuition and music; all forms of insurance; contributions, dues and gifts; street car fares; the barber; laundry; cleaning supplies and soap; toilet supplies; telephone; postage; moving; such servants' wages as were paid; automobiles, bicycles and trucks; moving expenses; liquor, taxes and tobacco; the undertaker; funerals; and last, but sometimes by no means least, expenditures for sickness and disability, including whatever was spent for dental service.

These final expenditures, for sickness and for dental service, are different from nearly all of the others in one important respect. The amount of money that will be required during a given year for food or clothing or most family expenses can be estimated fairly closely in advance. But the expenditure for the care of health cannot be. No one knows where sickness will strike next, nor when. A family may live comfortably for several years, saving a little annually, with only slight illnesses among its members. Then, unexpectedly, the breadwinner may get sick, and the family be faced not only with the expenses incurred by his illness but with the loss of daily income as well. Under these circumstances, the savings of several years may be wiped out in a few weeks. Let Johnny take diphtheria or Susan develop an earache, with the possibility of a mastoid operation, and the resultant

TABLE 3.—EXPENDITURES OF FAMILIES WHO ACTUALLY PURCHASED SOME DENTAL SERVICE

Income Group	Amount Spent	Per Cent of All Families Purchasing
$ 900	$ 8.93	22.3
900-$1,200	13.12	35.0
1,200- 1,500	15.53	44.7
1,500- 1,800	18.84	51.0
1,800- 2,100	20.44	55.3
2,100- 2,500	23.99	59.3
2,500- up	28.62	62.3
All incomes, aver.	$17.77	53.7 per cent spent nothing

expense may endanger the independence of the family.

The extraction of numerous teeth, with consequent necessity for an expensive plate, inlays in cavities where amalgam fillings are not desired and roentgen-ray examination and treatments may also bring financial calamity. It is indeed true that many such expenditures could be avoided, if people did not wait until the last moment to go to the dentist. For some time, public and private health agencies, and many advertisements of tooth pastes, mouth washes and toothbrushes have been urging the public to "go to the dentist regularly." The tabulations made by the United States Bureau of Labor Statistics tell the story of what some people actually did. Table 2 gives the amounts that families in the different income groups spent on dental service.

It will be seen that the expenditures for dental care of these 12,000 families averaged barely $8.25 in a year, less than $2 per person. Amazingly small the figures seem. What could one purchase for the average expenditure? Two prophylactic cleanings, perhaps; two or three fillings. It is probable that such sums were not expended on prophylaxis, but on emergent reparative and restorative service, and the average is misleading. Table 3 brings us nearer to a true picture by showing that more than 50 per cent of these families spent nothing at all for dental care.

But even these averages do not show true expenditures. Some of these families had very little dental work done during the year; on others, the burden fell more heavily. Just how great the variations were is shown in Table 4, which gives figures from a special tabulation, made by the Commissioner of Labor Statistics, of the expenditures of the 1,226 families, about 10 per cent of the whole group, who lived in New York, Philadelphia and Boston. Sixty-one per cent of these families spent nothing; the expenditures of those who did varied from under $15 to $250.

Study of the table shows how expenditures increase with income. In the $900-$1,200 groups, three-quarters of the families spent nothing for dental

TABLE 4.—EXPENDITURE FOR DENTAL CARE BY 1,226 FAMILIES IN NEW YORK, BOSTON AND PHILADELPHIA.*

Amount Spent for Dental Care	Family Income Under $900	$900 to $1,200	$1,200 to $1,500	$1,500 to $1,800	$1,800 to $2,100	$2,100 to $2,500	Over $2,500	Total Number of Families	Per Cent Spending Specified Amount
Nothing	24	197	268	137	75	33	14	748	61
0.01—9.99	1	42	70	48	30	17	6	214	17
$ 10— 29.99	1	22	41	55	31	16	17	183	15
$ 30— 49.99	..	4	11	13	5	5	3	41	03
$ 50— 99.99	..	2	7	4	6	8	6	33	03
$100—249.99	..	...	...	2	1	1	3	7	01
Total	26	267	397	259	148	80	49	1,226	100

*From the tabulations of the U. S. Bureau of Labor Statistics.

care and only 2 per cent expended over $30; while, of the families with incomes over $2,100, only 36 per cent spent nothing and 20 per cent spent more than $30. These figures indicate that people pay more for dental service as they can afford to pay more. Those in the lower levels undoubtedly needed as much dental work done as the more fortunate families in the upper income levels. But a small income, even when well managed, will stretch only to a certain point.

Another example of expenses for dentistry is given by Prof. Dwight Sanderson.[1] Out of seventy-six families in two villages, thirty-three reported expenditures for dental care amounting to $336, an average of $4.42 each. In two rural areas of the county in which there were 173 families, 116 families reported some dental service, amounting to $1,733.75, an average of $14.49 per family. The records are estimated to be incomplete by as much as 15 per cent, probably somewhat more than this. These farm families, like the laboring group, are evidently obtaining much less dental service than they need.

But not only wage-earning and farming groups have to wrestle with the finances of dental care. Prof. Jessica B. Peixotto in her recent book, "Getting and Spending at the Professional Standard of Living," analyzes the expenditures of ninety-six families of professors in the University of California, of whom 60 per cent were living upon less than $5,000 a year, the average being about $4,000.

She says:

> An inspection of the outlay of these families for dental care seems to bear out the presumption that lack of funds, not ignorance of the value of dental prophylaxis, forces neglect of proper precautions, for the amount spent on dentistry increases directly with the amount of total expenditure. The six families reporting no dentist bills were all living on amounts less than $5,000 and the six who spent less than $10 on dentistry are in the same income group. A total expenditure of $6,000 seems very definitely the dividing line. Below that income, from 50 per cent to 60 per cent of the families spent less than $50 a year; more than 80 per cent spent less than $100. The exception is the $5,000 to $6,000 group. Here only one third spent less than $50; 28 per cent spent $100 or more. This increase and emphasis is probably due to the presence of growing children in these families. When the total expenditure exceeds $6,000, the cases spending less than $50 practically disappear and more than half spent over $100. For the first time we see some families spending more than $300. It may be argued that the higher expenditure groups as contrasted with the higher income groups, for they are not identical, have a few more children and that these children are perhaps older and more in need of dental care. But inspection proves this to apply accurately only to the lowest group with expenditures below $3,000, who are all younger men with the average of only one child.

In a summary of her findings, Professor Peixotto comments further on expenditures for dentistry:

> From the data it seems fairly certain that these faculty families economize on preventive dental work because they must. In this they are probably representative of a large class of spenders in the world at large. All higher costs of dentistry appear in the higher expenditure levels only. From this fact it seems fair to argue that the lower costs of dentistry in the lower income groups may be due in part at least to inability to pay or to a relative unreadiness to allot surplus to dental prophylaxis.

It is a striking fact that more than 90 per cent of these professional families spent something for dental care and that the average family expenditure was $50. At the highest income level of the wage-earning group, only 62 per cent spent

1. Sanderson, Dwight: Study of Sickness in Rural Areas in Cortland County, New York, Cornell University publications, Memoir 112, March, 1928.

anything for dentistry and their average expenditure was less than $30. Obviously, the standard of living of the university families included more or less systematic attention to the teeth. Nevertheless, it is safe to say that a very small part of that $50 went for preventive work.

Statistics give at best a cold picture. Individual experiences with dental bills and family budgets are more vivid, though we must be cautious about regarding them as representative.

It is from middle-class families, whose standards of living tend to outrun their incomes, that tales of financial woe are most heard. Occasionally, one hears someone declare pridefully "I haven't spent over $15 a year on the dentist for the last ten years." On the other hand, I know of three persons with incomes ranging from $1,800 to $2,400 a year, who, within a few months, paid $285, $245 and $175, respectively, for dental care. In one case, this meant giving up a vacation entirely. There was a young couple with an income under $5,000 a year who found living in New York on less than that sum a considerable feat, and who spent in the first two and a half years of their married life nearly $600 for dentistry in addition to $75 or so for physician's services. This dental work included a minor operation following an extraction, poorly done, and several other extractions, as well as roentgenograms, fillings and a denture. In some cases, fees were reduced in their behalf. Even so, they felt the burden to be great.

A young woman earning less than $3,000 a year and spending a good proportion of it on the support of her mother paid out nearly $300 in less than two years for a partial denture (three teeth), prophylaxis and other necessary work. This would have been entirely beyond her means had not the dentist allowed her to pay him on the installment plan.

These are all people whose financial status is considerably above that of those studied by the Bureau of Labor Statistics. They would not be eligible to the ordinary dental clinic according to present standards. Yet bills like these are felt to be a severe strain by people who already have difficulty in maintaining themselves in accordance with inherited or desired standards. They recognize the value of dentistry, and even of preventive work. Table 5 presents a year's dental expenses of a family of five persons.

TABLE 5.—DENTAL EXPENSES OF FAMILY OF FIVE

Father		
Five amalgam fillings		$15.00
Mother		
Three amalgam fillings	$ 9.00	
Two roentgen-ray examinations	6.00	
One extraction	3.00	
One inlay	20.00	
		38.00
Children (3)		
Twelve amalgam fillings (an average of four each)	$36.00	
Two extractions of first teeth	6.00	
		42.00
		$95.00

Had this family added two prophylactic treatments for each of the three children and one each for the father and mother during the course of the year, the total would have been increased by $32. But this family, with an income of barely $3,500, was already spending much more than the average expenditure for dental care among families of this group. The extra amount for prophylaxis would have seemed an extravagance unless they had been convinced

that preventive work done regularly would keep down their bills in the future. It will be noticed that the rates paid the dentist ($3 per filling, etc.) are very moderate as city prices go. The dentist had advised that the eldest child have his teeth straightened, but the parents regretfully felt orthodontic work to be quite beyond their means.

The rates charged by dentists appear moderate or high to the patient in proportion to his means and according to his point of view. The patient does not readily appreciate that the dentist must depend for his living on his working time and that, according to what seems to be a usual estimate, about one-third of his income must go toward meeting his professional expenses, leaving only two-thirds of his gross earnings for himself and his family. The dentist who charges $6 for an amalgam filling, taking him half an hour, is charging at a rate (working 1,600 chair hours) that would yield him a net income of only $6,400.

It is always difficult for one person to understand sympathetically the other fellow's budget. A woman writes complaining bitterly of a dental bill for "One gold crown, $20.00, one gold inlay, $17.50, and four tiny fillings of alloys, $10.00. The bank check of $47.50 which I gave his (the dentist's) nurse was a *whole week's pay* for my husband." The patient's view necessarily depends on the relation between his expenditure and his income. Whether the layman who sits in the chair knows less about the dentist's finances than the dentist knows about his patient's may be a question. Certainly, the facts in this paper seem to indicate that the greatest limitation on the average dentist's earning power is the limited amount that can or will be spent by most families for dental care in competition with the innumerable other demands on the family budget.

It is common knowledge that greatly increased and more intelligent attention to the care of the teeth has been the rule in certain sections of our population of recent years. Some of the figures presented in the preceding pages have illustrated that the amount expended by a family for dental care increases even more in proportion to their intelligence than in proportion to their income.

How much can the spending for dental care for the average family be increased by education in the value and importance of good dental service? Certainly, much can and should be done in this direction. Nothing, obviously, is of more moment to the dentist. He faces, today, a vast untapped market for his services just as the lay public faces the problem of a vast amount of neglect of needed but unsecured dental services; unsecured in some localities because of the lack of a dentist, but much more generally because of lack of means.

I cannot escape the conclusion that present-day family budgets are in the main too low for adequate dental service, and even though some increase in dental expenditures may be brought about through education, for the mass of the population in this country, family incomes are not high enough to provide, at usual dental costs for high-grade work, all or nearly all the dentistry that is desirable. In particular is it true that preventive work is very unlikely to be bought on a private practice basis by the mass of the population. Emergency and reparative work will take precedence, and even if spending habits for dentistry can be generally shifted upward, they are not likely to include much if any preventive work for the mass of families. It is for this reason

that most public health workers and leading dentists everywhere have become convinced that, for children at least, preventive work must be provided, much on the same basis as is education.

I am not presenting the statistics and comments in this paper in criticism of a profession with many of whose members I have been associated for years and whose hard-won advances in standards and whose large and increasing public service can win only respect and appreciation from every informed person. Contact with dentistry as an interested and sympathetic outsider has, however, led me to feel it worth while to present some facts and points of view which are perhaps not so readily perceptible to those within the dental profession itself. Evidence of widespread interest in the economic side of health service is manifested by the recent organization of the national Committee on the Cost of Medical Care, with economists, doctors, dentists, nurses, public health officials, lay men and women as members; a group which affords opportunity for particular professions to cooperate in the broad studies which it is beginning.

It seems to me that dentists and the lay public together face two outstanding questions: 1. How much can family dental budgets be increased by education? 2. How much can the cost of dental service be reduced by planned organization of service or by various technical means? As to the first point, progress has been made in recent years, but much more is needed. Concerning the second question, there is much lay complaint that when planning or furnishing service dentists give too little consideration to the economic factor. Maximum economy in fillings, dentures and other services, so far as is consistent with efficiency, is the sound policy for dentists and public alike. Encouragement of luxury habits works in the long run to the disadvantage of both profession and public. In so complex and pregnant a situation, dentists should join forces with the general public in studying and dealing with the economic facts and factors on which the welfare of both so largely depend.

METALLOGRAPHY OF GOLD NICKEL ALLOYS

By JOHN S. SHELL, B.S., San Francisco, Calif.

THE series of alloys formed by melting various proportions of gold and nickel together have formerly been classified as forming a eutectic mixture with the approximation composition of 25 per cent nickel and 75 per cent gold.

In a recent article,[1] it has been shown that this conception of these alloys is erroneous in that a eutectic forms only within certain temperature ranges, while, under other conditions, a continuous series of solid solutions is obtained similar to the case of gold and copper. The diagram developed by Fraenkel and Stern is shown in Figure 1, which indicates that, through an interval of 200 C. or more after complete solidification, the alloys remain as solid solutions.

1. Fraenkel and Stern: Ztschr. anorg. & allg. Chem., 166:161, 1927.

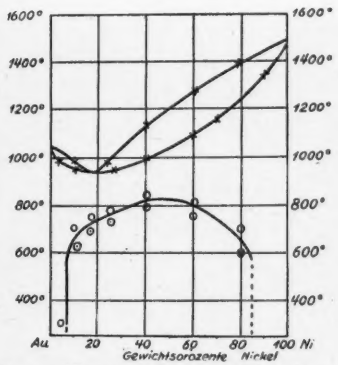


Fig. 1.—Temperature composition diagram of gold and nickel. (From Fraenkel and Stern.)

Fig. 2.—Gold nickel containing 5 per cent nickel quenched from solidification temperature. (× 30.)

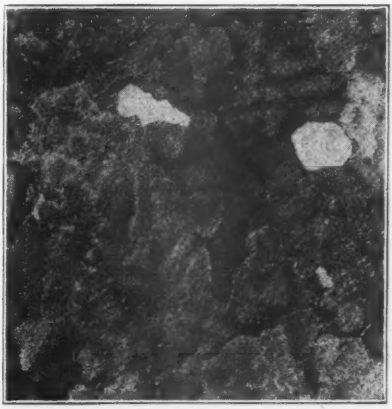


Fig. 3.—Gold nickel containing 5 per cent nickel cooled slowly. (× 30.)

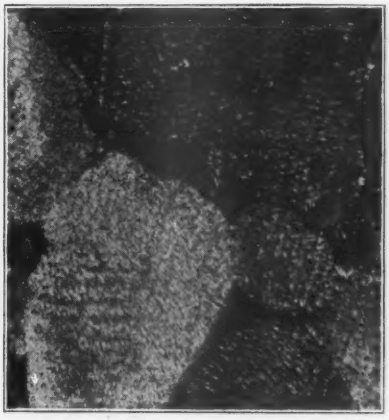


Fig. 4.—Gold nickel containing 5 per cent nickel cooled slowly. (× 150.)

An alloy containing 15 per cent nickel and 85 per cent gold heated above 1,000 C. is completely liquid. If the temperature is allowed to fall to about 950 C., solidification starts, and the temperature drops more slowly, during which time a continuous series of crystals containing a slightly increasing amount of nickel are formed. At 930 C., solidification is complete, and the metal should be homogeneous with the possible exception of some coring. As the temperature continues to drop, no change takes place until about 700 C. is reached. According to Fraenkel and Stern, at this point the alloy changes its structure and separates into two constituents, which constitute a eutectic mixture. This should modify the physical properties as well as the internal

Fig. 5.—Gold nickel containing 10 per cent nickel cooled slowly. (× 150.)

Fig. 7.—Gold nickel containing 15 per cent nickel quenched from red heat. (× 150.)

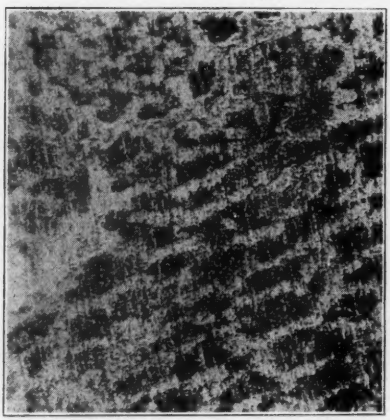


Fig. 6.—Gold nickel containing 15 per cent nickel quenched from solidification temperature. (× 150.)

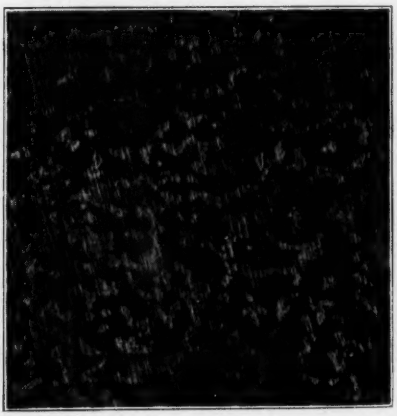


Fig. 8.—Gold nickel containing 15 per cent nickel cooled slowly. (× 150.)

structure, as such transformations are usually reciprocal.

Many of the dental alloys now in use contain various proportions of nickel, so that a correct casting and heat treating technic should be followed when any of these alloys are used, if optimum results are to be obtained. A dental casting containing nickel, when cast and quenched, would have a different internal structure and would probably differ considerably in physical properties from the same alloy when cast and allowed to cool slowly.

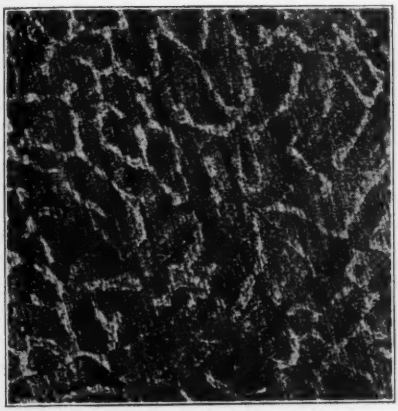


Fig. 9.—Gold nickel containing 20 per cent nickel quenched. (× 150.)

Fig. 11.—Proprietary alloy quenched (× 150.)

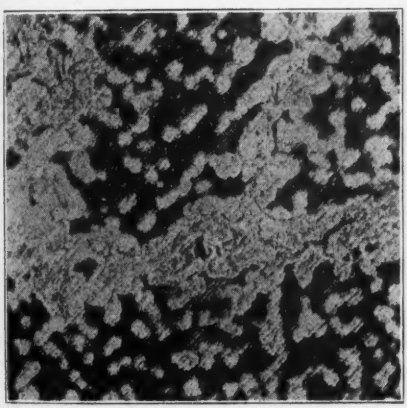


Fig. 10.—Gold nickel containing 20 per cent nickel cooled slowly. (× 150.)

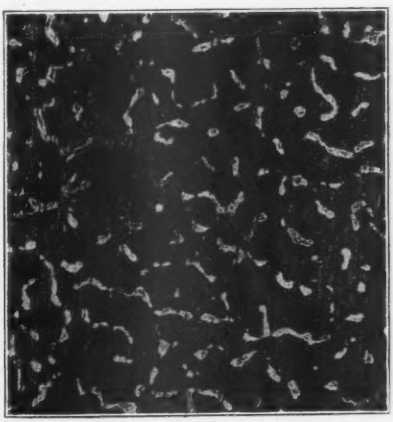


Fig. 12.—Proprietary alloy cooled slowly. (× 150.)

According to Figure 1, alloys containing less than 7 per cent nickel form solid solutions during the solidification range and do not show any transformations below the melting point. It seems possible that, with a sufficiently slow cooling rate, solid transformation would take place throughout the entire range of alloys rather than suddenly discontinuing at 7 per cent nickel.

Alloys of gold and nickel containing 5 per cent nickel, when slowly cooled through a period of from eight to ten hours, show a change in microstructure which may be a result of coring taking place during solidification or may be due

to solid transformation below the melting point. Figure 2 is a photomicrograph of a gold nickel alloy containing 5 per cent nickel which has been quenched as soon as it became completely solidified. It shows the typical dendrite structure of a solid solution with no trace of coring. Figure 3 shows the same alloy cooled slowly from the melting range to room temperature. The heterogeneous structure around the grain boundaries can probably be interpreted by assuming solid transformations which would be more rapid in the portion of the alloy containing the greatest proportion of nickel. Figure 4 shows the same section at increased magnification, which indicates the change to a eutectic structure more clearly. Figure 5 shows a section of a gold nickel alloy containing 10 per cent nickel cooled slowly, and the same transformation takes place, although more definitely, than in the alloy containing 5 per cent nickel. When quenched, this alloy shows a structure identical to Figure 2. A 15 per cent nickel alloy, even when quenched from the melting point, shows a similar type of segregation around the grain boundaries as shown in Figure 6. The most rapid quenching possible fails to eliminate the white structure surrounding a darker interior, and no section containing a homogeneous solid solution as in the former alloy has been obtained, although a sufficiently long annealing at a temperature slightly below the solidification range should eliminate all coring. This may be interpreted as due to the rapid rate of transformation in the solid state. Figure 7 shows the alloy more slowly cooled although the specimen was quenched above red heat. Somewhat more of the alloy has now been transformed to the new phase. Figure 8 shows a section of the alloy cooled in the furnace, and here the transformation seems to be almost complete. Figure 9 shows a section containing 20 per cent nickel, and regardless of the rate of cooling, the structure is not homogeneous.

This is again probably due to the rapid rate of transformation in the solid state and, in this case, not due to coring, as the solidification line in Figure 1 can be seen to pass through a minimum at 20 per cent nickel.

Figure 10 shows the same alloy cooled slowly in the furnace, and the structure indicates that the transformation is almost complete. Figure 11 shows a section of one of the dental alloys now being used extensively after being melted in a furnace and quenched from the temperature of complete solidification. This structure corresponds to those shown above.

If the alloy is cooled slowly from the melting range at room temperature, the structure changes in much the same manner as the binary gold nickel alloys, as is shown in Figure 12, although this alloy contains other ingredients.

THE VALUE OF GOLD FOIL IN MODERN PRACTICE*

By C. N. JOHNSON, D.D.S., Chicago, Illinois

IF the romance of dentistry is ever written, gold foil will be one of the leading characters. If the full story of dentistry's service to humanity is ever told, gold foil must be given the major credit for this service. If the palm is ever yielded to dentistry for the development of an unusual technical superiority over other professional callings, gold foil must be accorded the chief honor. If the source is sought for the outstanding achievement in saving the natural teeth after they are attacked by decay, gold foil must claim its full share in this beneficent function.

Thus, when any adequate consideration is given dentistry, gold foil must be acknowledged as a prominent factor in its development. Never has any one material so signally stamped its individuality and superiority on any calling as has gold foil on dentistry.

Then why are we today presented with the spectacle of the almost total eclipse of foil in the dental firmament? Why do we find men who admit, or rather who claim, that they never use gold foil, men who boast that they have long since discarded their gold pluggers? In view of the acknowledged service of foil in the past, the present tendency is something to be taken into consideration. There is little to be gained by blinking at a fact when it confronts us, and this is a fact of such proportion that we cannot ignore it. It is said that there is a reason for everything under the sun, and so there must be a reason for the change that has come over the profession in its attitude toward gold foil. Let us look somewhat carefully into this reason.

Is it due to the fact that gold foil has lost its virtue as a filling material? Is it not possible to do with foil today what the masters did with it years ago? Do the manufacturers not prepare their foil as well as they formerly did? I have on many occasions made the statement, which I now repeat, that there never has, in the alchemy of nature, been a material produced that will so surely save a tooth as a well adapted, well inserted, well condensed, well contoured and well finished gold foil filling. This fact holds as true today as ever it did. Foil has not lost its virtue, nor has the manufacturer failed us.

But something inevitable has entered into the situation, and whatever is inevitable must be reckoned with—it cannot be ignored. The introduction of inlay work, rightly or wrongly, came very near to sounding the death knell of foil. The only reason that it has survived at all is because of its intrinsic merit, which nothing in the way of allurement or propaganda could quite overshadow. In looking into the reasons

*Read before the Section on Operative Dentistry, Materia Medica and Therapeutics at the Sixty-Ninth Annual Session of the American Dental Association, Detroit, Mich., Oct. 26, 1927.

why inlay work came so near to eliminating foil from dental practice, we will discover several contributing factors, and at least one of these takes us outside the profession. No consideration of this subject is complete without facing that great sublime entity known as our patients; and it is hardly stretching the point to say that our patients summarily took the matter out of our hands, by waving foil aside and beckoning inlays in.

To my mind, the great outstanding limitation of foil is the hardship on the patient. This appeals to me far more than the plea of a hardship on the operator. I have never been much impressed with the hue and cry against gold foil on the theory that its use was too exacting on the operator. The very exactions accompanying its use have been the means of developing a skill among dentists which has brought to them the admiration of the world, and without which the profession would never have achieved what it has. Today, there is as great a need of skill as ever; and on this ground alone, if for no other reason, every dental college which aspires to teach students properly should demand as a requirement for graduation a large volume of gold foil operations. It is senseless to claim, as some are trying to do, that an adequate training in finger craft may be obtained from the technic of inlay work. It never has and it never will. There is something about the manipulation of gold foil that brings out the essence of expertness as does no other operation in dentistry. Those who shirk this inevitably lose something for which no amount of effort in other technical procedures can ever compensate. One does not have to stretch one's imagination very far to experience in the manipulation of gold foil a spiritual uplift which sanctifies the operator and makes of him a better dentist and a better man. I once heard a practitioner remark that every operator should insert at least one large gold foil filling at frequent intervals for the moral effect on him; and I am sure that those who have had the pleasure of using foil extensively will at once recognize the inference. To begin with, gold foil demands cleanliness, and this in itself is a supreme virtue in dental practice. Then, it demands exactness and precision, and these are also cardinal requisites for the best service. It demands concentration, and no great work is ever accomplished without concentration. As a source for training of the hand, eye and brain, there is nothing in dentistry to compare with this material, and no student is ever adequately equipped to go out to practice dentistry who has not had a thorough drilling in the manipulation of gold foil. Instead of being a hardship to the operator, the use of foil becomes one of the most delightful and pleasurable of all operations to the man who is expert in its manipulation.

Therefore, the contention is here made that we are not doing our duty by students unless we compel them to master the manipulation of foil. Even if, as some declare, these students, when they get out to practice, will use inlays to the exclusion of foil, the fact remains that the training they have had in the use of foil is one of the best means of preparation for technical procedures of any kind. There is no question that a student will be a better operator in every respect; he will make better inlays, better amalgam fillings, better crowns and bridges, he will prepare cavities better and do all kinds of operations with greater facility and precision if he

has had a thorough training in gold foil. If the manipulation of foil was not an important factor in an operator's ability, our state boards would not continue to demand it as a requisite for licensure. Every thinking man in the profession will agree that the boards are wise in making this one of the chief requirements, and it will be a retrogressive step if they ever abolish it.

There are two considerations which seem to me largely accountable for the change on the part of the profession from foil to inlays—the hardship on the patient when foil is employed and the saving of time with the inlay. Let us look the facts squarely in the face; there is no question that a foil operation of any size exacts of the patient a greater toll in the way of nervous energy—to say nothing of the actual pain — than does a similar operation with an inlay. There is no avoiding the issue, and no argument in this matter. A patient who has once had an inlay inserted will never submit to a long foil operation again; and probably you and I should feel the same if we were the patient. As far as the relief of tension is concerned, the inlay came as a distinct benefaction to people who had to have teeth filled, and this was so instantly recognized by every one that it became the chief factor in the landslide to inlays. Argue with people as we may about the greater permanence of foil over inlays, our argument falls on deaf ears when the tedium of a foil operation looms before them. They have tasted blood, and we cannot feed them a stone.

Not only this, but there is a decided saving of time to patient and operator in the inlay method. The sittings are shorter and much of the technical procedure may be done by an assistant or even by the operator himself in the absence of the patient. Altogether the process of inlay making is so devoid of stress compared with a foil filling, there is little wonder that it has made such a widespread appeal.

In addition to this, the operator may easily manage more cavities in a given number of sittings with inlays than with foil, and thus he can render more service in a stated period of time. He can care for more patients in a day because the sittings are shorter, and, in brief, he can turn out a greater aggregate amount of work because so much of it is done in the laboratory.

All of this, of course, applies to operations of appreciable size, and it is at this point that a vigorous protest must be made against the prevalent tendency on the part of many in the profession to resort to inlays in all classes of cavities regardless of size or condition. Not till our operators recognize the virtue in every process and accord to each method its legitimate field will the best service be meted out to the people whom we serve. It is just as much an aberration for an operator to discard gold in many of the smaller cavities as it would have been to ignore inlays when they were introduced and subject the patient to the tedium of a long and painful foil operation in the distoclusal surface of a second or third molar where half the crown must be restored. When we hark back to the days in which we made such operations in foil and recall the tension and discomfort involved, we are impressed with the utter folly of subjecting our patients today to such a procedure; and it is equally foolish to lean the other way and use inlays in small cavities where there is neither a saving of time nor stress, and where the result can never be as good as with foil.

Therefore, when we are called on to consider the value of gold foil in modern practice, we must insist on its use in all pit and fissure cavities wherever found, and as these constitute an appreciable percentage of the cavities occurring in the teeth, it will be seen what a large field there still remains where foil is clearly indicated. To use inlays in the cavities mentioned is not only ill advised but is also a waste of time. These cavities can be filled with foil in less time than inlays can be made for them, and there is no comparison in the results. To make inlays in many of these cases, it is necessary to cut the cavity much larger than for foil, and the technic for these small inlays is much more difficult than for large inlays, and certainly than for foil. In general, the smaller the cavity, the greater the indication for foil, and the larger the cavity, the greater the indication for inlays. There is another factor also which must be considered. Some patients are more manageable than others, and will submit to foil operations with better grace. A great deal hinges on this. If a patient is amenable to control, foil may be used in many cases in which otherwise it would be a mistake to attempt to use it. The day is past when dentists can afford to subject patients to an unreasonable amount of discomfort, or to unreasonably long sittings. The nerves of patients are more acutely tuned today than they were a decade ago, and this fact must be reckoned with. In this, we are confronted with a condition and not a theory. It is folly to attempt a long and exacting operation with a patient whose nerves are so highly strung that it is impossible to maintain any control, and the result is usually not only exhaustion to patient and operator but also a very indifferent operation.

The reaction to these things in our patients is changing, and while some operators with more magnetism than others are able to obtain better cooperation on the part of the patient, the fact remains that the rank and file will seek a dentist who does not subject them to too great discomfort.

In addition to the cavities mentioned, there is another class of cases in which as far as utility is concerned, gold foil has no rival. In proximal cavities in incisors, there is no other operation which will yield the permanent results that foil will. I have had the pleasure of seeing foil fillings in the proximal surfaces of incisors which had been doing good service for more than sixty years; and in these cases, there is no possible dream of the imagination that could have accorded to any other material this tenure of service. But here, again, we find ourselves confronted with an "if." If we can keep these fillings from showing, our patients will permit us to use them; otherwise, they will not. There are certain individuals in whose teeth the exposure of gold is not conspicuous or objectionable, and, in these cases, foil should be used. In the mouths of blonds, gold is seldom noticeable, and where utility is paramount to appearance, there can be no question of the advantage of gold over other materials.

Proximal cavities present something of a problem. If there is any place where gold inlays are unsatisfactory, it is in these cases. Any attempt to employ them usually results in imperfect margins unless the tooth is unduly opened into a large cavity. Porcelain inlays at one time promised a satisfactory solution, and many of them did good service; but if we are to judge of their utility by

the extent to which they are used today, we must conclude that they have been more or less disappointing. The silicates have had their advocates, and undoubtedly there is a place for them, although this place is very limited, and as far as permanence is concerned, they cannot be mentioned in the same breath with gold foil. In short, no other material has, up to this time, afforded us the same assurance of prolonged service in these cases as foil, and if the patient is more desirous of saving the teeth than of catering to esthetics, there is only one choice.

To summarize: The value of gold foil in modern practice is found in its application to such cases as pit and fissure cavities, to all cavities in the molars and bicuspids where access is not too difficult and where the tension is not too great on the patient—in other words, where a good technic may be obtained without undue stress—and to all cavities in the anterior teeth where esthetics may be sacrificed for utility and length of service.

With a full recognition of the exceptions to these cases, there still remains a wide field for the legitimate use of foil, and to ignore its use under its proper indications is to fall far short of the best service to the patient. As has already been said, there is no material as reliable as this, and to discard it because of its exactions or its few limitations, in the face of its manifest and manifold virtues, is either to ignore unlimited opportunity for the best service, or to shirk supinely an insistent and moral responsibility.

DISCUSSION

Perry F. Gilbreath, Kansas City, Mo.: No one can deny that the greatest piece of art, demanding the greatest skill in dental operations, is the gold foil filling. It has stood the test of time and, without question, is one of our most valuable assets as a filling material. It has developed a manipulative skill in cavity preparation and handling filling materials as nothing else has. The best operators that our profession has produced have used it extensively for over a half century. To condemn it or even to doubt its merits would almost be equal to destruction of the very foundation on which our present day conception of operative dentistry is built. Nevertheless, with all its superior characteristics of durability, technical training, tooth preservation, etc., that inevitable something has brought about a change in present-day operative procedure in restoring decayed teeth. Gold foil, as everyone will admit, has, to a large degree, been relegated to secondary place and, with many dentists, has been entirely discarded as a filling material. The reason for this condition Dr. Johnson has very plainly explained, and in his diplomatic manner he is considering the position of the dentist, and especially the man who is trying to render the public the most efficient service that dentistry can produce. He also views the situation from the patient's standpoint, which is very important. Dr. Johnson's years of experience as a practitioner, teacher and writer qualify him to speak plainly and to warn us against shirking our responsibility in conserving tooth structure and giving to those entrusted to our care the best service that we are capable of rendering. He states that there are two considerations causing the desertion of the foil filling, one the hardship on the patient in inserting foil gold, the other the saving of time, both for operator and patient, resulting from use of the inlay. I confess that in my own experience, these two factors have governed me in many decisions as to what I would use, even though the patient was willing for me to select the filling material that I considered best for the individual case. This especially applies to the proximal cavities in the anterior teeth and the pit and fissure cavities in the posterior part of the mouth. It is in these teeth that Dr. Johnson upholds the use of foil gold. His statements are clear and well founded when he says that the use of gold inlays in small or medium size pit and fissure cavities is not only ill advised, but also a great waste of time and loss of good tooth structure. Dr. Coston once said in discussing this subject, that a man with a very large practice was asked if he used gold inlays. He said that he did, then he was asked how he used them; he answered

"Exclusively." He then was asked how he used them in small fissure cavities, in bicuspids and molars. His reply was that he cut out the cavities until they were large enough to put an inlay in them. This is no doubt true of many practitioners, men who are conscientious about their work but have allowed themselves to fall into the procession, placing inlays in every cavity coming to their attention. Gold inlays hold a very important place. Many honest skilful men have spent hours developing a technic that has brought the inlay to its present high standard, where it saves many teeth that would otherwise be lost or the surrounding tissues impinged by the gold crown. I believe that, generally speaking, approximal cavities in the bicuspid and molars can be handled with a greater degree of satisfaction, to both operator and patient, by the gold inlay than by any other filling material, except where too much tooth structure is cut away for removal of the wax pattern. In that case, gold foil or amalgam is indicated, as also in the gingival third cavities in the same teeth, and, I might add, in very large occlusal cavities in the molars. Alloys, porcelain inlays and silicates have their place in restorations. It would be hard to practice dentistry without them. Just where the line should be drawn is the question. In considering the anterior teeth, esthetics has come to be an important factor. As long as there was no other material to fill these teeth with, our patients were contented with the gold filling, but since the introduction of porcelain and silicates, their wishes have to be considered. Just as these patients refuse to endure long foil operations, they likewise refuse to have the conspicuous gold in the front of their mouths. With all the argument that it is an inferior substitute, they still insist that a material be used that is in harmony with the color of the tooth, even though it is necessary to replace it at frequent intervals. I agree that the places for the present silicate materials are very limited and in no way can they be mentioned in the same breath with gold foil when it comes to permanency. I cannot refrain from raising the question: Are we who are engaged in the science of practicing and teaching health preservation giving our conscientious service or are we allowing the dental manufacturers and temperamental public to dictate what we should use in filling teeth? I appreciate the fact that the dental manufacturers have done a great deal for the dental profession in producing various materials that are most worthy. They no doubt have given their best efforts to develop a filling material that meets all the requirements, but until they succeed we should cling to that which we know is the nearest to the ideal. We cannot be guided entirely by any set rules or methods of procedures, but must be guided largely by circumstances and the exercise of our best judgment. If we are going to use the silicate materials, we should acquaint ourselves with their characteristics and develop a skill in their manipulation that will assure their greatest efficiency. Silicate, like the long abused amalgam, can never rank very high until an exacting technic is properly carried through. I agree regarding teaching dental students the technic of manipulating gold foil. It is vastly important that dental colleges continue the full measure of instruction in the use of gold foil and not diminish the number of such operations required of students. I believe most colleges are fulfiling these requirements by giving extensive training in placing gold foil fillings and are doing so at a great disadvantage in comparison to the time before the gold inlay and silicate materials. The dental student is not sufficiently interested in making gold foil fillings, largely because of the outside influence of the practicing dentist. With all the enthusiasm and pressure that is brought to bear from the Dental Educational Council, college officials, faculty members and infirmary demonstrators on the importance of this training, the student is still uncertain as to its necessity, otherwise than to make his grades. Dental students, by the time they have reached the senior year, have friends and acquaintances among dentists; some spend their vacation periods in dental offices, and it is only natural that they see and know what kind of operations are being done. The student visits other offices and discusses with the dentist gold foil fillings, and finds that there is no such thing in his office. The most distressing thing of all, the dentist is indifferent toward gold foil and refers to it as being out of date. The colleges have a great responsibility, but there is also a responsibility resting on the shoulders of every man practicing ethical dentistry. They owe it to these students to encourage them to take advantage of the opportunity to develop that skill in finger craft in making gold foil fillings by which they will increase their manipulative

skill and be better prepared for other more highly complicated technical procedures. I am glad that Dr. Johnson placed so much emphasis on college training. It not only stimulates those who are attempting to teach cavity preparation and gold foil operations, but should be an incentive to each and every dentist to cooperate with the college to perpetuate the art of manipulating the peer of all filling materials. The gold foil filling is by no means becoming extinct. There are many capable men throughout the country making foil fillings today, faithfully and loyally maintaining the high standard of their restorative operations and preserving the health of their patients' teeth. Too much credit cannot be given these men for their endeavors to uphold the high standard and teach others to do likewise. Quoting from Dr. Woodbury, "There is nothing about the manipulation of gold foil that cannot be acquired by any one whose natural gifts fit him to practice dentistry. With the mastery of gold foil technic, goes the desire to develop fully the ability to restore tooth form and function in all its phases." Gold foil has not lost its virtue. There is still a place for it. Some may ignore its use, but I feel that the dentist who is endeavoring to give his best service to his patient will continue to use gold foil fillings where they are indicated.

William Finn, Pittsburgh, Pa.: We all acknowledge that the gold foil filling, when properly made, is the *best* kind of filling, and one of the poorest when not properly made. I think the last part of that sentence presents one of the reasons why so many make inlays in nearly all cavities. It is because of a lack of technical ability so much needed in making these fillings that they are abandoned. This technic should be taught in the dental school, but we cannot expect the student to be a finished workman; it is this desire drilled into the student that makes him look for more knowledge, and hence, a study club where this technic is developed in its highest sense. I do not wish it to be understood that I would make gold foil filling in all cavities; that is impossible because of lack of access, the size of some cavities, the inability of the patient to stand malleting because of looseness of the teeth or ill health, the impossibility to adjust the rubber dam in such cavities as the gingival third, which are far below the gum tissue, and many other reasons. The lack of access is one of the chief reasons; there are many places where the line of force cannot be obtained. I do not quite agree that patients waived the foil aside and beckoned the inlay in. Of course, they seconded the motion, and had much to do with carrying it, but the operator is the one who made the motion and saw that it was carried out. *For what reason?*—lack of knowledge and ability, laziness and a lack of a desire to do the best for the patient.

CONSIDERATION OF MALOCCLUSION IN MUTILATED CASES*

By BURNE O. SIPPY, B.S., D.D.S., M.S., Chicago, Ill.

IN medical terminology, "mutilated" implies the deprival of an organ. This definition applied to orthodontia means, it is assumed, a malocclusion in which there is a deprival of a part of the denture or its surrounding and associated tissues. Our consideration will be limited, therefore, to dentures deprived of the possibility of their normal function by the absence of such tissues. It will be advisable to consider the possibilities under this limitation in order to group our material for careful consideration and to exclude such other material not falling within the scope of this study.

In the normal denture, there is occlusion with a full complement of teeth. In malocclusion, there may or may not be

*Read before the Section on Orthodontia at the Midwinter Clinic of the Chicago Dental Society, Jan. 25, 1928.

a full complement of teeth. Teeth may be unerupted or impacted—and a distinction is made between the two conditions—or there may be both unerupted and impacted teeth. A very large proportion of cases of malocclusion have this involvement, but these are outside our present consideration. In another group, there is not a full complement of teeth. There may be one or more missing for cause; namely, accident, extraction, congenitally, or as the result of surgical procedure, and these conditions will now be considered.

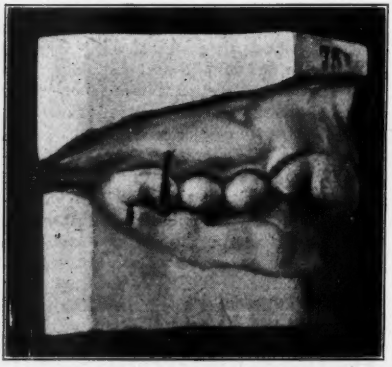


Fig. 1 (Case 1).—Original model, right view.

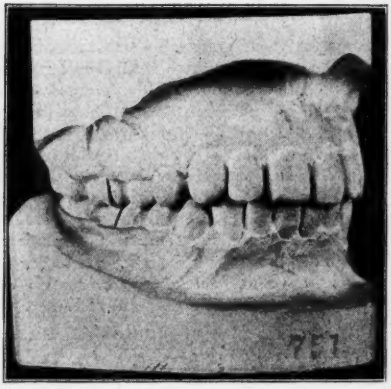


Fig. 2 (Case 1).—Retention model, right view.

Accidents to the deciduous denture, particularly in very young children, and the secondary damage to the developing permanent tooth germs are difficult to manage. Many times, nothing of value can be done, although frequently some form of temporary splint or a deciduous orthodontic appliance may be placed to correct the displacement of deciduous teeth and thereby give freedom to the otherwise cramped permanent teeth; which obviates malformation or malposition of the permanent teeth involved. In the premature loss of deciduous teeth, we find a regular and almost certain cause of malocclusion. This premature loss may be the result of neglect of caries, and the progress of destruction may warrant early extraction. Should this occur in the area where there is no developing permanent tooth soon to take its place, the mutilation is greatly increased. Not only do we find the usual malocclusion resulting from the premature loss of deciduous teeth, but also the lack of any development in the bone because of the absence of the corresponding or adjacent permanent tooth. In all such instances, it is not so much the loss of the temporary tooth but the failure to provide adequate artificial space retention that causes the damage to the developing denture. Even after all of the teeth are erupted, accidents occur that may remove the teeth completely or make their removal necessary. This is likewise necessary when a tooth is in the line of fracture of the maxilla or mandible.

There are two forms of extraction: the necessary, or unavoidable, and the pernicious. When the child comes to the dentist for the first time with a tooth so badly involved by caries that it is not

scientifically or humanly possible to save the tooth, it, of course, must be extracted. I desire to repeat "scientifically and humanly," because either or both may be overlooked, and teeth may be extracted because it is easier and less troublesome to all parties concerned for the moment. When such practice is employed, it is, of course, malicious. The other form is the pernicious extraction of a perfectly normal and healthy tooth to correct the crowded condition of certain teeth, which again may relieve the situation for the time being but inevitably produces serious consequences at a later date, perhaps years after. May I refer in passing to a study[1] which treats with the effects of the loss of the first permanent molar some years after extraction. The extraction of a bicuspid or a pair of bicuspids for the same reason is likewise pernicious.

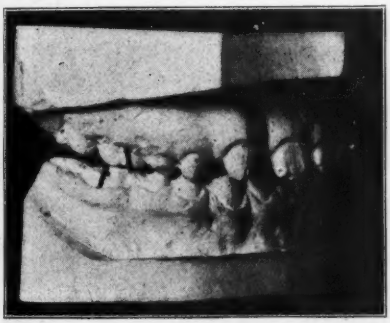


Fig. 3 (Case 3).—Original model, right view.

Fig. 4 (Case 3).—Original model, left view.

In regard to third molars, my position has been stated before, but it may be well to state briefly here that I believe that it is a mistake to extract third molars unless the surrounding tissue is involved in an infection; or they are hopelessly impacted and are active to the point of upsetting occlusion, or are increasing a malocclusion, or they are definitely the cause of referred pain. I am satisfied that third molars are many times diagnosed as impacted and surgically removed when, if given time and opportunity, they would have erupted and come to full occlusion. Wherever third molars have been so extracted needlessly and unnecessarily, it should be considered as a mutilation, as the normal denture is robbed of one, two, three or four molar teeth which Nature has provided and which go to make up the full complement of teeth. I do not believe that it can be said or proved to be a good thing for any denture to be mutilated to this extent without cause as just described.

Accidents to permanent teeth should be followed by immediate dental care or they should be kept under careful observation. Frequently, some form of simple splint will support a tooth that has been loosened by a blow and will relieve the pericemental membrane and supporting tissue of unnecessary and excessive strain during the period of reorganization of the tissues involved, or,

1. Sippy, B. O.: Effects of the Loss of the First Permanent Molar, J. A. D. A., 14: 1953 (Nov.) 1927.

many times, will prevent strangulation of the pulp. Where a permanent tooth has been lost completely by accident, some means of space retention should be employed at once, being continued until such time as one reaches the age when a permanent restoration may be made judiciously. This form of prevention of malocclusion is frequently overlooked, and unless it is afforded, there is usually some shifting of the teeth or failure of complete development of the arches. When the time for restoration arrives, a patchwork job results, which is always unsightly, inefficient and a continued source of annoyance, worry and trouble to the individual. For the same reasons, extraction of permanent teeth for cause, except third molars, should be followed by space retention in the developing denture and the space or spaces should be maintained until the full development of all teeth, except third molars, and the development of the arches has occurred, at which time the permanent restoration can be made with success and satisfaction. Otherwise, it is not possible. At this time, it should again be said that the old theory unfortunately accepted by so many, that should the permanent molar or molars be lost for any cause, the second molar will move forward taking the place of the lost first molar and the third molar will move forward and take the place of the second molar is, as I hope that I have helped to prove, not a scientific statement, as in only a very small percentage of cases, under the most favorable circumstances, is this true. The larger percentage of cases develop malocclusion of one form or another, which is serious to a degree comparable to the time of extraction in respect to the developing denture and the extent of the disturbance in normal development prior to that time.

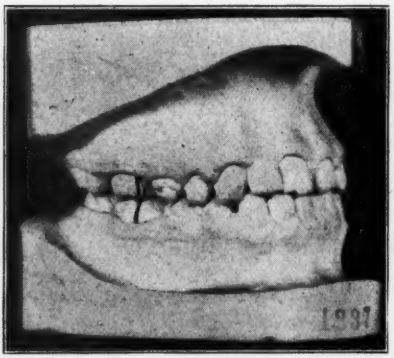


Fig. 5 (Case 3).—Final model, right view.

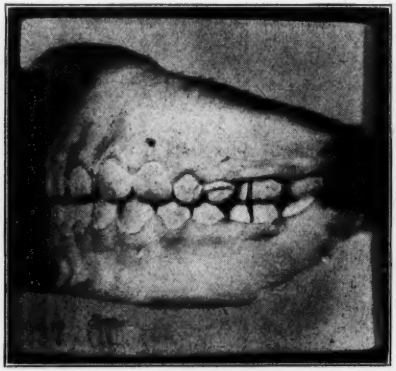


Fig. 6 (Case 3).—Final model, left view.

As far as is known, there is no satisfactory scientific explanation for the congenital lack of teeth. Those who are inclined to believe in hereditary influences may include such influences as causative in this group. In a paper by Merle C. Coulter[2] of the University of Chicago and presented before the Chicago Association of Orthodontists, the closing statement is: "Could any such phenomenon (as heredity and environment) affect the tooth or jaw tissue? I think

2. Coulter, M. C.: Heredity and Environment, J. A. D. A., 15:1860 (Oct.) 1928.

not. Immunologists tell us that the mammal will manufacture antibodies for only two of its own tissues, lens and spermatozoa; hence, it seems inconsistent with the best scientific knowledge to believe that by this or any other means inheritance of acquired tooth or jaw characters could be effected."

After surgical procedure in cleft palate operation or the removal of a tumor involving the bones of the face and jaws or when a considerable infection is present and a large sequestrum removed, it is usual to lose one or more teeth and

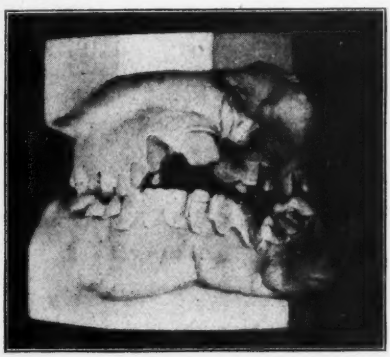


Fig. 7 (Case 6).—Original model, front view.

perhaps other tissues associated with the denture. With the better surgical technic, the postoperative results are less serious and less damaging, but it is seldom that one or more tooth germs are not unavoidably destroyed. In a paper read at a meeting of the Chicago Association of Orthodontists, B. Lewis,[3] of the University of Illinois staff, recently of the Speech Defect Clinic of Vienna, states that our surgical technic in cleft palate and harelip operations far surpasses that of Europe. It is found particularly difficult to correct the speech defects in these patients operated on abroad because of the very imperfect soft palate or the lack of a soft palate that is usual; whereas, in this country, there is little difficulty in the correction of these cases because of the care employed in preserving the soft tissues. B. Lewis was particularly enthusiastic over the results obtained in speech correction in patients operated on for cleft palate.

A surgeon, in performing plastic operations, often requires the assistance of an orthodontist. I have been for-

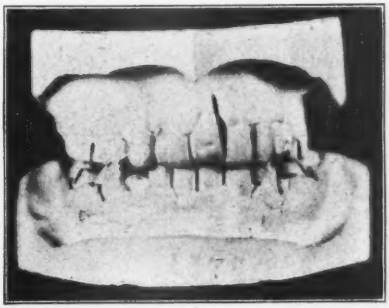


Fig. 8 (Case 6).—Final model, front view.

tunate in being called on to assist surgeons in such operations on several occasions. Four cases of different types of mutilation will be cited, showing the methods employed. In assisting a plastic surgeon, the orthodontist meets conditions that are quite different; consequently, the plan of the operation must be understood in order to determine how to be of the greatest assistance in the surgical procedure.

The treatment of mutilated cases of malocclusion is difficult and at times unsatisfactory. Usually, the longer the tooth, teeth or tissues have been missing, the more difficult the treatment and the restorations that are to follow. A digest of a questionnaire which B. Frank Gray

3. Lewis, B.: Speech Defects, J. A. D. A., 15:1867 (Oct.) 1928.

of San Francisco assembled under the title "Further Study in Compensatory Treatment for Missing Teeth" would seem to indicate that the closing of spaces or the attempt to close the spaces resulting from the loss of teeth is in favor and is the rule rather than the exception. It should be remembered that the report was based upon the statements of the fifty-three men who responded to the questionnaire. There are many others whose opinions are not included in the list and without a complete response one should hesitate to draw conclusions.

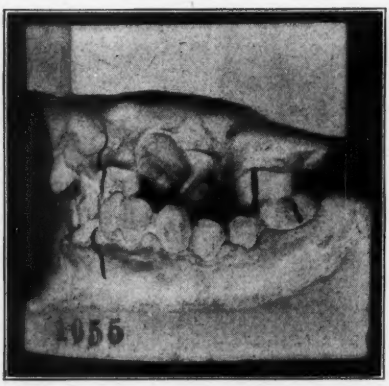


Fig. 9 (Case 7).—Original model, left view.

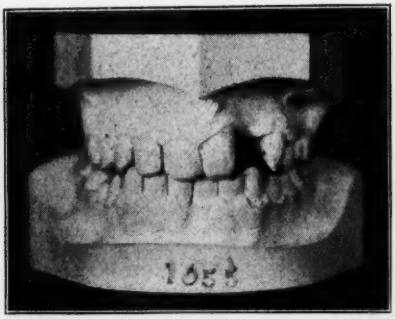


Fig. 10 (Case 7).—Period of treatment model, front view.

CONCLUSION

It is a mistake to follow any set rule while dealing with human tissues, and yet there are principles which can safely be followed. The principle of restoring occlusion and maintaining a full complement of teeth in mutilated cases by artificial means is a safe and sane procedure and in the end the patient profits greatly by such management. It is my practice to maintain spaces where teeth are recently lost or to establish the normal space of the missing teeth when the mutilation has been of long standing. One must use reason, as there are times when such management is impossible or impractical; but this is the exception rather than the rule. The management of mutilated cases is as varied as the mutilations. It is as difficult as the extent of the mutilation. It is folly to attempt the impossible, but there is satisfaction in preserving as much of the mutilated face and jaws as is possible.

It should be remembered in making a diagnosis or a case analysis, in outlining a treatment plan and in formulating a prognosis that a complete case history, full mouth and, if possible, extra-oral roentgenograms, orthodontia casts, or a face mask or both, and photographs are essential. The general features should be carefully studied to get as broad a view of the conditions as possible; and then the specific details can be worked out more accurately. The classification of the malocclusion should be determined and should be the principal basis of the treatment plan as a mutilated case cannot be treated successfully without being treated completely as to its classification. By establishing or maintaining normal mesiodistal and buccolingual relations and establishing normal inclined planes of the teeth present, the problem of the mutilation is reduced to solution.

REPORT OF CASES

CASE 1.—Class II, Division II (Mutilated).—*History.*—The tonsils were large but apparently not infected. At 9 years of age, the patient was pushed against a drinking fountain, breaking the crown of the right lower central incisor, but not exposing the pulp, which subsequently died. An abscess developed and the tooth was extracted. No effort at space retention was made. The models and roentgenograms showed that the space of the missing tooth was partly closed.

Treatment.—There were two periods of treatment. In the first period, the active management of the case covered three months. Retention for the class of case was established. The space was maintained by a temporary bridge for three and a half years, when a permanent bridge was constructed and set. The patient then moved to Cleveland, where he remained for one year.

In the second period, active management was maintained for seven months; retention, for mesiodistal relation only, for twelve months. The total period of active management was for ten months.

Treatment plan: The normal bilateral mesiodistal relation of the arches was established by tipping the upper molars distally and the lower molars mesially, correcting the relation of the long axis of the upper central incisors by tipping the incisal edge labially, depressing the lower anterior teeth and obtaining a little greater width in both arches in the first bicuspid region. The space for the missing lower right central incisor was to be maintained.

Retention plan: The normal mesiodistal relations were held by intermolar bite guides, using a split anchor band on the lower first permanent molars with spurs soldered to the buccal surfaces of these bands engaging the mesiobuccal surfaces of the upper first permanent molars at their mesiobuccal angles. Also intercuspid bite guides were necessary, using plain bands on the lower cuspids with cast guides which engaged the mesiobuccal angles of the upper cuspids. A temporary bridge consisting of plain gold and platinum orthodontic bands on the lower right central and lower left lateral incisors, with a lower left central porcelain crown facing ground to fit, backed in the usual way and soldered to the bands, was cemented to place and worn for three and one half years, after which time a permanent bridge was constructed.

Fig. 11 (Case 8).—Face mask, front view.

Fig. 12 (Case 8).—Left view of appliance used, showing splints on all teeth present, and intermaxillary high temper spring.

CASE 2.—Class I, Median Line Involved and Mutilated.—*History.*—The lower left first molar was extracted at 6½ years of age. The patient had scarlet fever, chicken pox and measles prior to 6 years of age. The tonsils and adenoids were removed at 12 years. The patient was presented for orthodontic treatment at 9½ years.

Treatment.—There were two periods of treatment. During the first period, active management was continued for four mouths. For retention, a lower vulcanite retainer was constructed with a spacer for the missing tooth and with an amalgam occlusal surface to antagonize the upper left first molar, rebuilt from time to time for three and one half years.

In the second period, active management was maintained for five months. Retention of space for the missing tooth was maintained for four months. Total active management was maintained for nine months. Occlusal restoration of all first molars and a permanent bridge for the space of the missing molar was made.

Treatment plan: The median line was corrected by moving the lower right second and first bicuspids to contact with the lower right cuspid, then all lower anterior teeth were moved forward and to the left, the lower right bicuspids being carried along until normal incline plane relations and the normal median line were established. Also the upper right first permanent molar, which was in supraclusion caused by the lack of resistance from its lower fellow, which was extracted, was depressed and the normal mesiodistal relation on the left side was maintained.

Retention plan: A lower lingual vulcanite retainer was constructed with a saddle at the lower right first permanent molar, having the amalgam occlusal portion built to engage the upper molar with normal incline plane relations. This was rebuilt from time to time and was always high, thereby creating occlusal pressure. The distal portion of this space retainer was fashioned to be as nearly as possible like the lower first permanent molar on the opposite side in size and contour, to produce as far as possible normal conditions in order to guide the lower right second molar to its normal mesiodistal position on its eruption. No other management was required to accomplish this purpose. The wearing of this space retainer for three and one-half years caused no discomfort to the patient and no damage to the hard or soft tissues of the mouth occurred. After this period, the occlusal surfaces of the three first permanent molars, which were never completely calcified, were restored, and the missing molar was restored by a removable bridge.

CASE 3.—Class II, Division I, Subdivision (Right Side), Mutilated.—*History.*—The grandfather had both upper lateral incisors missing. The mother had an extra cuspid. The child resembled the mother. The tonsils and adenoids were removed at 12 years of age. One tooth was broken at the incisal edge by a fall while the patient was young. She was not physically strong. Orthodontic treatment was sought when the patient was 15 years old. The roots, as revealed by the roentgenograms, were in such position that the plan of treatment was to use the upper right cuspid as the right cuspid and the upper left cuspid as the upper left lateral incisor, opening spaces for the upper left lateral incisor and right cuspid and lower cuspids.

Fig. 13 (Case 9).—Face mask model before operation.

Treatment.—There was but one period of treatment. Active management was maintained for fourteen months. For permanent retention, a removable gold prosthetic piece bearing the two porcelain teeth (replacing the upper left lateral incisor and the upper right cuspid) was made.

Treatment plan: The normal mesiodistal relation on the right side was established by tipping the upper right molars distally, likewise the upper right bicuspids, and by tipping the lower molars mesially, also the usual intermaxillary ligature management was employed on the right side; the normal mesiodistal relation maintained on the left side; the crown of the upper right cuspid moved distally to contact with the upper right first bicuspid, i.e., its normal place in the denture, opening space for the missing upper right lateral incisor; the crown of the upper left cuspid moved mesially to a point of contact with the upper left central incisor, the cuspid being thereby substituted for the lateral incisor on the left side and space opened for the upper left cuspid.

Fig. 14 (Case 9).—Appliance used in plastic operation for the restoration of the chin and the floor of the mouth.

Retention plan: The mesiodistal relation was held on the right side by the intermolar bite guide (as before described) and a vulcanite plate retainer bearing porcelain vulcanite teeth constructed to supply the missing ones and maintain the spaces until such time as the permanent restoration could be made.

Case 4.—Class II, Division I, Mutilated.—*History.*—The patient had a double mastoiditis at 17 months of age. The tonsils and adenoids were removed at 7 years. The mother and grandmother had congenitally missing teeth. The case was presented for orthodontic management when the patient was 12 years old. The models and roentgenograms showed that the upper third molars, the lower lateral incisors and the lower right second molar were missing; also that the lower cuspids were congenitally missing, and spacing was equal.

Treatment.—There were two periods of treatment. During the first period, active management was maintained for nine months. Retention (spaces maintained) was continued (twelve months) until the upper cuspids erupted.

During the second period, active management was maintained for four months. Retention to maintain the lower cuspids and central incisors in normal relation has continued six months to date. A space retainer was placed until the time of permanent restoration. Total active management was for twenty-one months. Retention time totaled eighteen months plus.

Treatment plan: It is planned to correct the mesiodistal relation by use of intermaxillary ligatures; to close the upper anterior spaces and change long axis of the upper central and lateral incisors; to move the lower central incisors mesially to contact and to median line with the upper central; to move the lower cuspids distally to normal contacts, thereby opening normal spaces for missing lower lateral incisors. This may be done as the spaces of all four lower anteriors are in favorable position and the crown portion of these teeth have tipped because of the lack of resistance of the missing lateral incisors.

Retention plan: It is planned to retain according to the class of the case, with bracket bands on lower cuspids and lateral incisors and a ribbon arch segment lugged distally to the central brackets and mesially of the cuspid brackets until permanent restoration of the missing teeth may be made.

Case 5.—Class I, Mutilated.—*History.*—No personal or family history was involved. The patient was presented for orthodontic treatment at 11½ years of age. The models and roentgenograms showed all upper bicuspids to be missing, with prolonged retention of the deciduous upper cuspids. These roots were absorbed, but the crowns maintained their normal positions and the upper cuspids had erupted distally to their normal position,

taking the position of the missing bicuspids and thereby following the line of least resistance.

Treatment.—The periods of treatment were three, with probably another in prospect. During the first period, active management was maintained for seven months (the patient leaving for Europe). Retention of space was continued for eight months.

In the second period, active management was continued for two months (the patient leaving for a trip west). Retention of space maintainers was continued for seven months.

In the third period, active management was maintained for five months (the patient leaving for Florida). Retention of space maintainers has continued for three months, to date. Total active management to date has covered fourteen months. To complete management at the present rate of progress will probably require one or two more periods of treatment. Prognosis is favorable, if the family will stay at home long enough.

Treatment plan: It is planned to extract the upper left temporary cuspid; move both upper cuspids bodily through the bone as the roots are in such position that they will not permit of the tipping of the crown mesially, disregarding the relation of the roots, and preserve the upper second temporary molars as long as possible, as there are no bicuspids to take their place.

Retention plan: It is planned to maintain the position of the upper cuspids gained during interrupted periods of management by a vulcanite palate bearing a .030 round gold-platinum wire contoured to fit the distal surfaces of these cuspids at the gingival margin and as much mesial tension as feasible. At the waxing state, the anterior portion of the palate is built up as a step for the lower anteriors to rest on when the jaws are closed. This step is to be made to establish what should be (as far as can be determined) the normal overbite for the individual. This is particularly important in view of the missing bicuspids and should be maintained until the eruption of the second permanent molar; otherwise, the first permanent molar will not be able to maintain the normal bite.

Case 6.—Class I, Mutilated.—*History.*—The tonsils and adenoids were removed when the patient was a baby. Operation for cleft palate and harelip was performed at the age of 2 or 3 weeks. Subsequent operations were performed, the last at 8 years of age. The patient was presented for orthodontic treatment at 16 years of age. The models and roentgenograms showed the upper left lateral incisor and third molar; the upper right lateral incisor, cuspid, bicuspids and third molar, and the lower second bicuspids and third molars to be missing. There were eleven teeth missing, i.e., seven congenitally absent and four absent probably as a result of the cleft palate operation.

Treatment.—There were two periods of treatment. During the first period, active management was maintained for sixteen months. Retention was maintained by a plate bearing the teeth from the upper left central incisor to the first permanent molar, for eighteen months.

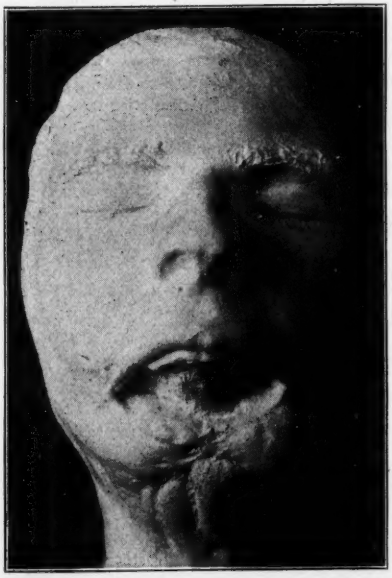


Fig. 15 (Case 9).—Front view after plastic surgery operation on chin.

In the second period, active management was continued for three months. Retention was maintained by a palate plate with necessary teeth (permanent). Total active management continued for nineteen months; intermediate retention for eighteen months.

Treatment plan: The upper arch was expanded mesially to the first permanent mo-

lars, to rotate the upper left second molar. The upper central incisors and right cuspid were moved incisally and to the right, this establishing the median line with the lower central teeth. The upper left first molar was moved bucally, bodily, to a normal buccolingual relation. Space was maintained for the missing lower left second bicuspid (after removal of the second temporary molar as it could not be saved) but space was not opened for the missing lower right second bicuspid.

It was advisable to take advantage of the compensation on the right side; namely, the

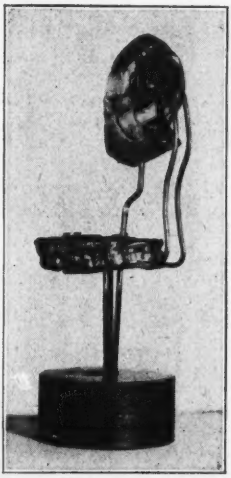


Fig. 16.—Appliance used in connection with plastic operation to form new upper lip by the advancement method, consisting of upper full metal splint (cast) with removable attachment arch to which was soldered two parallel wires, ends of which were soldered to a swaged metal piece fitting the forehead and bridge of the nose. Adhesive tape was stretched over these wires and affixed to the advanced flaps of the cheeks, forming the new upper lip. (Case referred by Dr. Phemister, of the Presbyterian Hospital.)

missing upper right lateral incisor and lower second bicuspid.

Retention plan: A full upper vulcanite palate retainer bearing a left lateral cuspid and first cuspid was placed; the labial portion of the palate extended well up under the lips and was of such contour as to fill out the lip where needed because of lack of bone

CASE 7.—Class II, Division II, Subdivision (Left Side), Mutilated.—*History.*—A cleft palate and harelip operation was performed when the patient was 3 months old. Subsequent operations were performed at 2, 3 and 6 years of age. Correction was made of the lip and also of the nose at 10 years of age. The tonsil tissue was used in the palate operations. No nasal history was obtained. The cleft involvement was particu-

Fig. 17.—Appliance used as a nasal prop in transplantation of cartilage to the septum of the nose. The upper splint was first cemented. The removable part was placed at the time of the operation, which was performed by Drs. Gilmer and McArthur.

larly difficult. The patient was presented for orthodontic treatment at 12½ years of age. The models and roentgenograms showed that the upper right lateral incisor was missing.

Treatment.—There were two periods of treatment. During the first period, active management continued for sixteen months. Retention was established by a vulcanite palate retainer, to maintain the width of the arches, and bearing a porcelain tooth. This was continued for twenty months.

In the second period, active management continued for nine months. Retention (as before) has been maintained for six months to date. Dr. Moorehead made the final correction of the lip and closed a small opening in the palate. Total active management was for twenty-five months; retention, for twenty-six months. Further treatment is not anticipated, and permanent restoration may be

made at the conclusion of the retention period in from three to six months hence.

Treatment plan: As the two right first molars represent the only occlusion in this denture, general expansion of the entire upper arch is needed. It is planned to correct median line, which is to the left, to the extent of the full width of the upper central incisor; to correct the mesiodistal relations in the left side by moving the upper right first molar distally; to open and maintain the space of missing upper left lateral incisor; to depress the lower anterior teeth and provide room for the eruption of the lower second bicuspids, particularly the lower right second bicuspid, which is impacted.

Retention plan: It is planned to maintain the general expansion in the upper arch by a full vulcanite palate retainer bearing a vulcanite tooth at the upper left lateral incisor; to maintain the expansion of the lower arch by plain anchor bands on the lower first molars with a lingual "G" wire soldered to the screw posts of the anchor bands and adapted to fit the lingual surfaces of the teeth resting above the cingulum on the lower anterior teeth.

CASE 8.—(Referred by Dr. Moorehead from the Presbyterian Hospital.)—*History.*—The patient was operated on for epithelioma, the operation involving removal of the right maxillary sinus and upper right maxilla. There were no teeth in the upper arch except the upper left bicuspids and first and second molars and no teeth in the lower arch distal to the first bicuspid as a result of the operation, and the extensive scar tissue ankylosis of the right temporomandibular joint followed. A subsequent operation was performed establishing a surgical joint on the right side midway between the symphysis and the angle of the mandible. The result was a very slight motion. The oral orifice was very small.

Treatment.—To assist in the establishment of a functioning joint, an appliance was constructed consisting of a cast splint covering the surface of the teeth present in the upper arch and likewise one for the teeth present in the lower arch. Lugs were soldered to either splint, to take a long high tempered steel wire with a fulcrum point, to give intermaxillary tension and to force the joint to operate beyond what was possible by muscular effort alone.

After eight or nine months of this management, sufficient excursion of the surgical joint was produced to enable the patient to bite an apple. From that point, it was only necessary to continue manipulation of the mandible by muscular effort. In time, there was a laxity of effort and a part of what had been accomplished was lost.

CASE 9.—(Referred by Mrs. Dean Lewis and Moorehead of the Presbyterian Hospital.)—*History.*—The face had been pinched between two railroad cars with resultant loss of the mandible and all teeth anterior to the first permanent molars. The two lateral fragments of the mandible collapsed toward the sagittal plane on healing of the original wound and original surgical efforts. There was no chin, and practically no floor to the oral cavity. Saliva drooled from the mouth in quantities and articulation was impossible. Eating gruels and drinking liquids were even difficult.

Proposed Surgery.—It was proposed to construct a chin from the tissues of the neck. Because of no support from the mandible, it was necessary that there be some form of support provided over which the tissue could be formed. Accordingly, an appliance was constructed consisting of a full upper cast splint with four half round sleeves soldered, the openings being parallel and directed alike. Four nickel silver wires of large gage were contoured as required. To the ends of these wires the arms fitting the before mentioned sleeves were soldered. A plate was then swaged to copy the contour and form of the anterior part of the mandible and bound by round wire soldered to its edge to prevent irritation. This was soldered to the four free ends of the four wires. This formed a removable part which, when in place, was rigid and substituted for the mandible. Before the plastic operation was performed by Dr. Lewis at the Presbyterian Hospital, the splint was cemented to place. At the conclusion of the operation, before the final sutures were placed the removable part was placed, and then the final suturing at the angles of the mouth was completed. The result was a somewhat more agreeable facial appearance, cessation of continual flow of saliva and comfortable eating (of soft foods) and drinking. The next step in the rebuilding process was the bone transplantation.

30 North Michigan Ave.

DISCUSSION

H. D. Swain, Kewanee, Ill.: Dr. Sippy has clearly and concisely covered the subject of mutilated cases of malocclusion. The paper contains much of value not only to the orthodontist but also to the general practitioner, who, many times, is directly responsible for the mutilation which these cases exhibit. The excellence of this paper results not only from its value as a treatise on the treatment of these mutilated cases, but also in its indirect appeal to the profession to minimize this condition of mutilation by better dental care. Dr. Sippy shows that here again, as in other types of malocclusion, a little prevention is worth much orthodontic cure. After defining and limiting the field of discussion by differentiating between incomplete cases of malocclusion (resulting from impactions and lack of eruption) and mutilated cases (resulting from loss of teeth), Dr. Sippy proceeds to emphasize the necessity of maintaining the physiologic proportions of the deciduous arch in either a natural or an artificial manner. The orthodontist is only too familiar with the tremendous importance to normal occlusion of preventing the premature loss of any part of the deciduous denture, for he is constantly dealing with the evils arising therefrom. Far too many parents and dentists, however, still fail to realize this, and Dr. Sippy's remarks point the way to a fruitful field for dental missionary work. The parents' only concern, oftentimes, is whether the neglected tooth will be replaced by a permanent one, and they entirely miss the essential point that it is not so much the loss of the tooth itself, bad as that is, which matters, but the loss of the space it occupies, which is worse. The dentist's position is somewhat different. Obviously, the solution of this problem of maintaining the deciduous denture intact during its normal retention is not to be found in the field of operative dentistry, but rather in the field of diet, nutrition, calcium equilibrium, prenatal care, etc. Until such results are obtained, the dentist must do, as Dr. Sippy says, whatever is scientifically and humanly possible to save these teeth or to surely retain their spaces and thus help eliminate a prolific source of mutilation. Dr. Sippy next deals with the complications resulting from the extraction of permanent teeth. The drifting, the mesial and distal tipping, the loss of interproximal contact, with the resulting soft tissue involvement arising from the loss of teeth, with no provision for space retention, are familiar enough conditions to need little comment. The remedy is obvious: permanent space retention. The fallacy of extracting teeth from crowded arches in the hope of obtaining an esthetic advantage with no functional loss has long since been established. In regard to the actual treatment of these cases, the principle of establishing and maintaining normal buccolingual, mesiodistal and inclined plan relationships while maintaining a full complement of teeth (in mutilated cases by artificial means) is, of course, the ideal. It is not always practicable or even possible to attain, but is always the goal to be striven for.

NEW METHOD AND TECHNIC OF FILLING ROOT CANALS*

By J. P. BUCKLEY, Ph.G., D.D.S., F.A.C.D., Hollywood, Calif.

IN this brief paper, I shall not discuss pathology or treatment except to say that, for twenty-five years or more, three rather general methods of treatment for dead and gangrenous pulp conditions have been more or less closely followed by the profession: the sodium potassium ionization method of Rhein; the sulphuric acid method of Callahan, and my own formocresol-phenolsulphonic acid method. About ten years ago, Howe introduced the silver-reduction method. Each of us, especially the first three named, have practiced and taught his

*Read before Odontographic Society of Chicago, April 5, 1928.

own technic. Because of its simplicity, the lack of time required and the results obtained, I still advocate and use formocresol for the initial treatment, followed by a thorough opening and cleansing of all canals with phenolsulphonic acid and broaches—the chemicomechanical process, as I call it. The younger men in the profession have selected from the various methods in vogue that which seemed to meet the demands in their practice. Much credit, therefore, is due such men as Coolidge and Blayney of Chicago, Hall of Kansas City, Grove of St. Paul, Best of Minneapolis, Johnson of Atlanta, and others, for from them we have learned that no one method need be followed in detail for successful results.

The surgery of root canal treatment has been developed today to such a high degree that little else is needed along this line; but, in my judgment, there is much room for improvement in the materials used for filling the canals after the root and bone surgery. The urgency of the demand and the genius of our profession will surely solve this phase of the pulpless tooth problem.

With this in mind for some years now, I, with others, have been endeavoring to supply this much needed improvement in our root canal work.

Tonight I appear before you with a new method and technic for filling the canals of pulpless teeth; and since my return to the practice of certain special fields in dentistry, this method has given me more satisfaction than any other I have ever used. I am happy to present it to you for your earnest and critical consideration.

Four or five years ago, I developed a material called dentinoid for protecting the dental pulp and filling the canals of pulpless teeth. This product served its purpose nicely for pulp protection, but for root canal work it lacked two essential properties: it was not sufficiently plastic to be readily introduced into a canal and it was not sufficiently radiopaque for all practical purposes. Within the past two years, I have succeeded in changing the formula of the original product so that now the new material, called dentinoid-radiopacic, meets every demand required in both pulp protection and root canal work.

The new dentinoid or dentinoid-radiopacic is a purely physical mixture of calcium phosphate and barium sulphate, with such antiseptics as thymol, thymol iodid and orthoform, and with a sufficient amount of resin which acts as a binder and gives plasticity to the mass when the powder is mixed with alcohol or chloroform. When thus mixed, it gradually hardens into a compact solid. Dentinoid is a medicated artificial dentin, a nonconductor, absolutely nonirritating, and is as nearly permanently antiseptic, though I hesitate to use the expression, as a material of this kind can possibly be. The antiseptics used are insoluble in water and are stimulating in character; and when the product is used for pulp protection in deep-seated cavities, it not only sterilizes the dentin immediately over the pulp, but also encourages the deposition of secondary dentin by stimulating the odontoblastic layer of cells, Nature's method of protection. When the product is used for filling root canals, it likewise keeps the canal and dentin sterile and tends to stimulate the cementoblasts, which, it is believed, will in time completely cover the foramina as the subsequent lamellae of the cementum are formed, when again we have Nature's protection. Subsequent radiographic interpretation seems to substantiate this view.

I shall not discuss the indications or contraindications for saving a pulpless tooth, nor shall I discuss the therapeutics or surgery involved in preparing the canal for the final filling, though in passing I should emphasize the importance of this phase of the work, for we should never attempt to fill any canal which has not been explored and properly opened. While the lead point used in the technic that I shall describe is far more stiff and rigid than a guttapercha point, it is not expected to go to place in a canal which has not been properly prepared for filling. This point cannot be emphasized too strongly. Unless we are willing to spend the time to do this work well, it should not be undertaken at all. Carelessness in this regard is what has caused the difficulty in the past.

I shall now describe in detail the mixing of dentinoid for use and the technic of filling the canal by the new method. There are only a few precautions to be observed in mixing the powder and liquid. A long series of experiments have shown that, for satisfactory results, the binder must be present in the powder in a certain and definite proportion; and, in the mixing process, it should be remembered that alcohol and chloroform freely dissolve both the binding substance (resin) and antiseptics. These liquids are thin in character and flow easily, and if care is not taken in making the mix, a certain amount of the binder and antiseptics will be removed by the selected liquid flowing through the powder and spreading over the slab, which alters the properties of the product and changes the formula to this extent. This must be avoided. In mixing, therefore, the desired amount of powder is placed on the slab and a concavity is made in the center into which the liquid may be added by drops from a small glass medicine dropper, the powder being incorporated as the liquid is thus added. It is not necessary to spatulate the mix more than just enough for the desired consistency in convenient handling and to bring the varnish, formed by the binder dissolving in the liquid, in contact with the insoluble bulk of the powder (calcium phosphate and barium sulphate). More powder or liquid may be added, if necessary, to accomplish this end, without danger of destroying the virtue of the product when set. The mix sets very slowly, and because of this property and its other physical characteristics, there is no need for haste when using the material for any purpose. Keep in mind that dentinoid is a purely physical mixture before and after mixing; it is not a chemical compound or a dental cement.

When filling the canals of pulpless teeth, alcohol should always be the liquid used for making the mix, which should be quite plastic. It is presumed that alcohol is the liquid last used in the canal for removing the surface moisture, which is all that is necessary when this method is followed. Some of the alcohol thus used is purposely left in the canal, for better results are obtained when the canal is slightly moistened with this liquid. With a suitable instrument, the plastic dentinoid is carried to the mouth of the canal and gently worked to the apical end, the operator filling the canal full and being certain that all air has been displaced. A lead point, which approximates the size of the canal, having been previously selected and tried in, and which, if desired, may also be used as a "diagnostic wire," is coated with the mixed dentinoid on the slab and then gently but firmly inserted in the canal as far as it will go. The point acts as a

piston or plunger, and, if properly selected, will nearly reach the apical end, forcing the plastic material into such multiple foramina and lateral openings as may be present and thus filling all space not occupied by the lead itself. When first fitted, the lead point should be marked and cut off about 3 mm. above the mouth of the canal, and this portion flattened with a pair of pliers. It should then be kept in an alcohol bath for final sterilization until ready to be coated with the dentinoid and placed in the canal. The flattened end facilitates handling and also permits the projecting portion to be easily bent against the floor of the pulp chamber. More plastic dentinoid is now placed over the point, and pressure is made with a small roll of cotton or bibulous paper. When it is known in advance that a post or dowel is to be subsequently fitted in the canal for some necessary restoration, the lead point should be shortened accordingly and only the apical third filled.

If for any reason the operator should prefer to use them, guttapercha points, ivory or other material instead of lead may be employed in practically the same way, except that with guttapercha, the selected point or points may be dipped in euchapercha compound, eucalyptol compound or chloroform instead of alcohol just before being coated with the plastic dentinoid and placed in the canal. After the point or points are thus placed, heat may be applied and the cones firmly packed.

In clean cases, as in pulp removal with no periapical involvement, or if no opening has been made by pathologic process or instrumentation, it is best to have the point stop just short of the end with the dentinoid beyond. But if there is an opening of any size, however made, the lead or guttapercha point should fit the opening; and, with this in mind in preparing the canal for the reception of the point, it should be made choke-bored. Thus, when the point is finally introduced, it will close the opening or stop where desired with very little, if any, of the plastic dentinoid being forced in the apical area.

Many dentists today are using both infiltration and conduction anesthesia, as indicated, for the painless removal of the dental pulp. In all such cases, in which asepsis has been observed in the operation, immediate root filling with dentinoid is especially indicated and advised, and the filling should stop at the dentino-cemental junction. If all details are carried out, absolutely no soreness follows. In cases in which the canal is so small that a lead or guttapercha point cannot be used to advantage in combination with dentinoid, the entire canal may be filled with the latter material with every confidence of success, and with the new product the root filling will be definitely radiopaque. If possible, the canal had better be enlarged to admit the selected point. If dentinoid is used alone, it should be packed as directed for filling the canals of deciduous teeth.

Dentinoid is the ideal material for filling the canals of deciduous teeth. In these cases, the entire canal may be filled with the material alone, which should be placed to the end and compressed firmly with bibulous paper or a small cotton roll. When the canal is thus filled, it should be covered at once with zinc oxychlorid cement and the cavity in the tooth filled at this or a subsequent sitting.

CONCLUSIONS

The practice of dentistry today requires that every effort be made to save the tooth pulp and keep it in a healthy condition. For safety and protection,

dentinoid should be placed in every deep-seated cavity immediately over the pulp; thus practically replacing the material that has been destroyed by the carious process. This certainly is better than the placing of some questionable foreign material, especially when it is remembered that the artificial dentin contains stimulating antiseptics.

Dentinoid adheres firmly to every surface of the tooth structure with which it comes in contact; but should it become necessary for any reason to soften the material in a cavity or canal of a tooth, this may be done by simply adding the liquid used in making the mix. This is a distinct advantage which a purely physical mixture has over a chemical combination.

In using dentinoid for filling the canals of pulpless teeth, it should be remembered that the material will become stiff as soon as the liquid is permitted to evaporate, but that it requires a few days before the product sets or hardens into a compact solid. In cases in which the root filling need not be disturbed for a post or dowel in some contemplated reconstruction work, it makes no difference about the delayed setting of the dentinoid, as long as it is known that it will ultimately set without shrinkage under the conditions and become extremely hard and resistant. When it is desired to use a post before the dentinoid sets firmly, only the apical half or third of the canal need be filled, and this covered with zinc oxychlorid or other quick-setting cement.

The several advantages of the combination of dentinoid and lead or guttapercha for filling the canals of pulpless teeth must be obvious. With this method, the dentin need not be entirely free of moisture; the removal of the surface moisture is all that is necessary. Some of the alcohol used for removing the surface moisture is purposely left in the canal, which in itself is an advantage. Then, too, alcohol has a great affinity for water, and the liquids mix in all proportions. This is an advantage in that the alcohol used in making the mix with dentinoid holds in solution the binder and antiseptics, which are carried into the tubuli when the plastic material is placed in the canal. Thus, the tubules are sealed with antiseptics therein contained, and the foramina and any possible lateral opening or openings are sealed with dentinoid, a hard, firmly adherent, medicated, artificial dentin, which aids rather than hinders Nature in the healing process. It seems to me that here we are approaching the ideal. Callahan, Groves, Puterbaugh and others have stressed the importance of sealing the tubuli with a varnish or resin solution.

The advantage of using the lead point with dentinoid in canals with large apical openings need not be emphasized; and the ease and certainty with which all canals can be perfectly filled to the end, and there only, by this method gives one a satisfaction not heretofore experienced. The recently published statistics of Blayney[1] emphasize again the importance of a technic that is certain and definite. With guttapercha and the solvents necessary to be used by the former methods in vogue, more or less guesswork was encountered even by our most careful operators. With the technic herewith presented, guesswork is entirely eliminated, and the results in every case, if the canal is properly prepared for filling, are positive and certain. This cannot be said of any other method with which I am familiar.

---

1. Blayney, J. R.: Chicago Dent. Soc. Bull., Feb. 24, 1928.

If I can read correctly the signs of the times and properly interpret the literature of medicine and dentistry, both professions are today admitting the overenthusiasm of some of their members in the recent past. Many physicians themselves have fallen victims to the craze for looking for focal infection in the mouth, and are demanding caution in the care of their patients. Dentistry is on the alert; everywhere men are investigating and studying the problem as never before; and the dentist of tomorrow who is not devoting all of his time to special practice must either learn how to treat the pulpless tooth correctly or turn this part of his work over to those who will do it right.

In making these statements, let it be understood that I am not advocating the treatment and retention in the jaw of all pulpless teeth regardless of how badly diseased or without consideration of the patient's general health. Nowhere in dental practice is there greater need for the use of those two essential remedies of which I have spoken so frequently in the past: tincture of good judgment and fluid extract of common sense.

DISCUSSION

J. R. Blayney, Chicago, Ill.: I can speak only in generalities because I have had no experience in the use of this material. I was pleased with the warning that Dr. Buckley gave that carelessness in the use and insertion of any filling material will unfailingly give poor results. This is the keynote in the search for a new root canal filling material. Many are in search of a material which is easy to place, the ease of operation appearing to be the sole motive for this search. It is perfectly permissible to have a material which is easy to work, but that must not be the guiding factor. Baseplate guttapercha, which most of us use, has certain limitations. It is not what I would call an ideal filling material, although I believe it to be the best at this time. From what Dr. Buckley says about his material, I presume it approaches much more nearly the ideal than base-plate guttapercha. One thing concerning the use of materials which are of a fluid nature when placed, and most of the new root canal fillings have this property, is this: As far as my experience goes, we are very liable to obtain too high a percentage of overfillings. The material certainly is radiopaque; that is important, yet it is not the sole purpose. I often say that we do not fill a canal for the purpose of taking a roentgenogram and exhibiting it to our patient. The only reason it should be radiopaque is that we may have some idea as to where the filling material went. Dr. Buckley spoke concerning filling canals immediately after pulp removal. It has been my custom to have at least one sitting intervene. Working in the mouth, we are compelled to seal the operative field immediately. I believe this is one of the few places in surgery where the operator hermetically seals the operative wound. If it were not for the contamination that would immediately take place, I do not believe that this would be good practice in pulp work; but when we are forced to choose between two evils, it is necessary for us to choose the lesser. After operative trauma, such as pulp removal, an inflammatory process ensues, and, to my mind, it is at least a safeguard to have that tooth in such condition that it may be easily opened, so as to ventilate the canal and to remove the inflammatory exudates that may be present. If they are excessive and the case has been permanently sealed, we have placed an additional burden on the tissues by expecting them to absorb all of the exudate. Dr. Buckley said something about a moist canal. I think that he referred to the natural moisture within the dentin. For fear that that might have been misinterpreted, I would like to suggest that canals that are moist from an inflammatory edema of the tissues surrounding the root end are not in a condition to be permanently filled. I have never been interested in a root canal filling material that could be placed while the canal was moist, because I never felt that the periapical tissue was in the proper condition for the final sealing of the tooth. I am encouraged by the results that have been shown here, and I should like the privilege of making some clinical tests.

Edgar D. Coolidge, Chicago, Ill.: I cannot judge the material, of course, without having had time to use it and examine it more fully. I cannot fully appreciate what has been brought to us, but it seems very hopeful and very encouraging. I feel thankful for having had the opportunity to see it as its first presentation.

From the very beginning of Dr. Buckley's teaching, with which I have had experience, he has been one of the most ardent advocates of cleanliness, asepsis and care. I believe that those things have more to do with the success of our work than anything else. He has always recommended the use of thymol, and I do not believe that we have as a profession really appreciated the value of this substance as an antiseptic. In a 1:2,000 solution, it is claimed that it will destroy all the bacteria found in the root canal in thirty seconds. And yet it does not destroy tissue. It seems to me that Dr. Buckley is consistently and persistently making use of some of the best principles known in treatment.

It is gratifying to me to see fillings from Dr. Buckley's early practice and from his recent practice. The results are encouraging.

TISSUE CHANGES IN TREATED TEETH OF KNOWN HISTORY*

By O. H. MOEN, D.D.S., M.S.D., Watertown, Wis.

IN presenting a paper on tissue changes in treated teeth, it is necessary to discuss these changes essentially from a histologic point of view. This, however, bears a direct relationship to oral surgery in the matter of diagnosis. The reaching out for some tangible evidence that will assist us in determining whether or not a tooth should be retained in the mouth has led to a great deal of research in that direction. Most of this has been and is being done in the field of bacteriology, although the work from a histologic point of view has increased during the past few years.

When we look at a roentgen-ray film and see a large area of involvement at the apex of a tooth, as registered by the ray, it is usually not difficult to give a diagnosis as to what should be done with that tooth. Our opinions as to the diagnosis may vary, but each individual usually has definite ideas concerning the retention or extraction of such a tooth. But when we view the roentgen-ray picture of a tooth, supported by a pericemental membrane that from various angles appears to be normal, or possibly slightly thickened, there are more of us who are in doubt as to what the diagnosis should be. So it is with the object of describing and showing the tissues found in root-filled teeth, with various degrees of apical pericemental membrane involvement, that this paper is offered. If it will in any way help us to visualize the probable histologic appearance of the apical tissues of teeth that we hereafter view on the roentgen-ray film, it will have served its purpose.

The material on which the data in this paper are based consists of twenty-five root-filled teeth which were extracted in my office during a period of a year and a half. The only teeth used are those of which a complete history as to root canal operations and treatment has been obtained. They are from the work of several practitioners and offer a va-

*Read before the Section on Oral Surgery at the Midwinter Clinic of the Chicago Dental Society, Jan. 26, 1928.

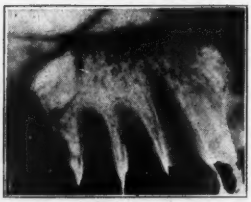


Fig. 1.—Upper cuspid of G. F., aged 30, retained six years after treatment with cocain pressure anesthesia and Black's 1-2-3. A gingival gold foil was placed in November, 1915. Recurrence of caries necessitated pulp removal in October, 1919. The tooth was extracted in August, 1925, as a possible factor in ulcers of the stomach. The pericemental membrane is normal, with root filling extending to within 1 mm. of the apex.

Fig. 3.—Lower second molar of E. J., aged 28, retained eleven years after treatment with procain pressure anesthesia and formocresol, after involvement by caries. It was extracted because the patient insisted on having no nonvital teeth. This tooth tested as vital at points ten and twelve on the "vitalitester." The pericemental membrane is normal and the canal filling extends to within about 1 mm. of the apex, although not filling the diameter of the canal.

Fig. 2.—Three millimeters from apex; good technic. *A*, accessory canals nearly closed by secondary dentin; *H*, hypertrophic degenerating tissue in a longitudinal section of an accessory canal leaving the main canal; G, guttapercha. (From tooth shown in Figure 1.)

riety of conditions. These histories and the roentgen-ray film of the tooth taken prior to extraction are used to help build a complete picture as to cause and effect

Fig. 4.—About 2 mm. from the apex. *T*, atrophic degenerating tissue; *G*, guttapercha point in this issue; *S*, secondary dentin following previous absorption *(X)*. (From tooth shown in Figure 3.)

of conditions found in the apical portions of the tooth, and which are shown in histologic sections made of each tooth. All of the histologic work was done, with the assistance of a technician, in the laboratories of Northwestern University Dental School, Chicago, under the supervision of E. H. Hatton and W. G. Skillen.

Several charts showing various relationships between treatment and results were made, but owing to the small number of teeth used, the data could not offer any definite conclusions. Some interesting facts, however, were noted. For instance, a chart showing the relation between completeness of root filling and the roentgen-ray appearance of the

apical tissues produced the following results: 1. The average distance from the apex of the root to the apical end of the root filling in ten teeth in which the roentgen-ray showed a normal pericemental membrane was 2.4 mm. 2. In five teeth showing a pericemental thickening, the average distance was 4.6 mm. 3. In ten teeth showing large areas of apical involvement, the average distance was 2.3 mm. The distances not occupied by root filling in the class having a normal apical pericemental membrane (2.4 mm.) and the class having large areas of apical involvement (2.3 mm.) are so nearly identical that it is impossible to make any inference as to the results of complete or incomplete root filling. It will also be demonstrated that a roentgen-ray picture showing a root filling extending to the end of a root has no great significance because it frequently fails to show whether or not the complete diameter of the canal has been filled.

Fig. 5.—Lower second molar of O. H., aged 57, retained three years after treatment by cocain pressure anesthesia and formocresol. The pulp was removed to relieve sensitiveness of the occlusal dentin due to abrasion. It was extracted, in full mouth extraction, with pyorrhea involvement, considered as a possible factor in ulcers of the stomach. The canal was filled. The apical pericemental membrane is normal, with the canal filling extending to the apex in one canal and about 2 mm. from the apex in the other, although irregularly packed.

GENERAL HISTOLOGIC FINDINGS

In the histologic study of sections of these teeth, the general observations were similar to those made by Skillen[1]. The prevalence of pulp tissue remaining after supposed pulp "removal" was the most noticeable condition. (The teeth in Figures 3, 8, 12 and 16 are marked illustrations of this condition.) It was also evident in nearly all the photomicrographs that the root-filling material failed to fill the complete diameter of even the accessible canals, to say nothing of the innumerable irregularities in the canals, which were invariably unfilled. (Figs. 2 and 7.) Many of these unfilled areas contained either débris as results of canal broaching or blood clots or remaining pulp tissue, the latter frequently in direct contact with the canal filling. (Fig. 11.)

TYPES OF TISSUE DEGENERATION FOUND

In all cases showing a normal, and in some showing a thickened, cemental membrane in the roentgen-ray film, this remaining pulp tissue was found to be undergoing one of the two general types of degeneration, an atropic fibroid or a hypertrophic fibroid.[1] In the atrophic degeneration, there is a "shrinkage or disappearance of the ground substance and many of the fibers" (Figs. 4 and 11), and in the hypertrophic, "an increase both in amount and density of the fibrous element." (Fig. 9.) Various degrees of both forms of degeneration may be seen in the same canal (Fig. 11), evidently due to various degrees of irritation, and both seem to result in a fairly stable tissue. In the cases in which the roentgen-ray showed an apical area of involvement, a hyaline degeneration with

1. Skillen, W. G.: Status of the Treated Tooth, J. A. D. A., 13: 291 (March) 1926.

no evident cell outline seemed to be the most prevalent.

FAILURE OF ROOT-FILLING TO SEAL CANAL

The most noticeable fact in all the cases with reference to the apical termination of the canal was that in not one was the foramen "sealed" by the canal filling. A condition of this kind might be expected in multirooted teeth, but it was also true of the single rooted teeth, and in successful cases as well as in failures. This would seem to conform to the statements that complete root fillings are impossible *in situ*.[2] We must therefore rely on the ability of the apical and periapical tissues to readjust themselves to their lessened function if favorable results are to be expected.

COMPARISON OF THE ROENTGEN-RAY AND MICROSCOPIC APPEARANCE OF ROOT FILLINGS

Many of these teeth, when judged from the roentgen-ray film, would be classed as good root fillings; but when studied histologically, they are shown to be poor. (Figure 1 shows the only tooth studied which shows good technic under both classifications.) This poor technic is most frequently due to the presence of unremoved pulp tissue. Guttapercha points extending several millimeters into vital tissue remaining in the canals, with

Fig. 6.—Apex of root; *G*, guttapercha point and dentin partitions in one main canal; *O*, other main canal empty with some absorption; *A*, accessory canals containing hypertrophic degenerating tissue with much secondary dentin. (From tooth shown in Figure 5.)

2. Grove, C. J.: Some Causes for Periapical Infections, with the Pathology, J.N.D.A., 6:679 (Aug.) 1919. Crane, A. B.: Pulpless Tooth Problem, J. A. D. A., 13:183 (Feb.) 1926.

this tissue closely approximating and surrounding the points and remaining vital in spite of this irritation, furnish an example of that fact. (Fig. 13.)

ritation (and infection), this remaining tissue will adapt itself to its lessened function and will not immediately break down as has been the more or less prev-

Fig. 7.—Two canals connected by narrow irregular channel; calcific degeneration and mummified tissue near filled canal (*F*). (The canal filling had fallen out in preparation.) *M*, mummified tissue in connecting channel; *H*, hypertrophic degenerating tissue, and *S*, secondary dentin with later absorption (*X*) in unfilled canal; *X*, absorption, with no secondary dentin in filled canal.

RESISTING POWERS OF UNREMOVED PULP TISSUE

The fact that many roots with poorly filled canals show no roentgen-ray evidence of apical changes, and that many of these, on histologic examination, show remnants of pulp tissue in various stages of degeneration, remaining in the unfilled portion of the canals, might lead us to conclude that, in the absence of irritation (and infection), this remaining tissue will adapt itself to its lessened function and will not immediately break down as has been the more or less prevalent idea, but will retain its vitality. It certainly does prove that the resisting powers of the pulp are greater than those with which it has generally been credited.[1]

And this is the point we want to remember: The tissues remaining in the apical portion of root canals, in teeth supported by a pericemental membrane that is normal, are not dead tissues,

neither are they undergoing decomposition by putrefaction. They are vital tissues, and this can frequently be demonstrated by anyone with the aid of an electric vitality tester. Many times, a so-called devitalized tooth, with a root filling that may have been in place for years, will give a positive response when tested by an electric tester.

The process of reconstruction of apical tissues that appears to take place was well described by Dr. Hatton[3] in 1926. He summarized the conditions activated by root-canal treatment and root fillings, in cases of favorable results, in three stages or phases: (1) the destructive or tearing down stage; (2) the rebuilding stage, and (3) the terminal or resting stage of the tissues.

The first phase, the destructive stage, is characterized by a rapid resorption of necrotic soft tissue and of all absorbable débris, and a considerable resorption of the walls of the unfilled portion of the pulp canal. This resorption is produced by an invagination of blood vessels and adjacent tissues from the apex or pulp stump and is further effected by the activity of either fixed or wandering phagocytic and resorbing cells. These activities are profoundly influenced by the type of treatment used, and it seems that either instrumental or chemical injury to these apical cells inhibits both this preliminary cleaning out of the apical field and the subsequent reconstruction.

The second stage, that of reconstruction, consists of covering over the denuded surfaces of the canal with a layer of calcified material resembling osteoid cementum, with typical lacunae and canaliculi. This layer is not of tubular structure and has no relationship to dentin. The filling material stump is covered by a layer of either fibrous or calcified tissue and sometimes both. . . .

After this calcifying process has come to a standstill, the third or resting stage begins. This lasts for years with but little alteration, except for a gradual fibrous atrophy of the soft tissues of the stump lying within the spaces of the unfilled portion of the canal.[3]

After considering these processes of reconstruction, it is evident that favorable results from canal treatment are more likely to be found where a minimum amount of tissue is left for reconstruction, rather than the enormous amounts frequently found, which is demonstrated in the photomicrographs here-

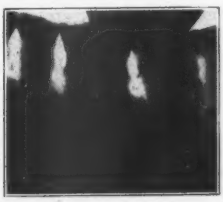


Fig. 8.—Lower second bicuspid of O. H., aged 57. It was retained three years after treatment by procain pressure anesthesia and formocresol. The pulp was removed to relieve sensitiveness of the dentin due to abrasion. It was extracted for reasons stated under Figure 5. The canal had been filled. The apical pericemental membrane is normal, with the canal point extending to within 5 mm. of the apex.

with. Certainly, the closer this tissue that needs to undergo reconstruction is to its blood supply at the apex, the more favorable the prognosis. Rarely are the canals of a tooth cleaned thoroughly, with all tissue removed, and yet frequently we find a normal pericemental membrane supporting a tooth which supposedly has the pulp removed, but where actually most of the pulp in the canals is still remaining. With favorable results accompanying the haphazard pulp removal of the past, the root canal operators of today, in the light of their present knowledge, and with the various aids now at their disposal, should produce results far superior to the majority of those in the past.

3. Hatton, E. H.; Skillen, W. G., and Moen, O. H.: Histologic Findings in Teeth with Treated and Filled Root Canals, J. A. D. A., 15: 56 (Jan.) 1928.

DANGER OF INJURY TO PERICEMENTAL MEMBRANE

We must not, however, become overzealous in our attempts to produce a perfect condition for reconstruction. Such overzealousness may lead to undue injury to the apical and periapical tissues.[4] From the specimens studied, it seems probable that injury to the latter tissues is more likely to prevent a normal reconstruction of the apical area than is the inability to remove all the pulp tissue. A normal degeneration of this remaining pulp tissue proceeds gradually and results in establishment of a fairly stable condition; whereas, irritation of the pericemental membrane soon sets up an inflammation of that tissue. This inflammation may lead on to suppuration,

Fig. 9.—At the apex, just above the lateral termination of the canal. *H,* hypertrophic degenerating tissue; *S,* secondary dentin; *N,* nerve trunk; *S,* secondary dentin extending to the root periphery in the wall of the curving canal just prior to ending on the lateral surface of the root; *X,* absorption. (From tooth shown in Figure 8.)

4. Puterbaugh, P. G.: Pulp Canal Therapeutics, J. A. D. A., 13:1384 (Oct.) 1926.

Fig. 10.—Upper second molar of G. F., aged 30. It was retained nine years after treatment by cocain pressure anesthesia and a mummifier. There was no attempt made to remove the pulp from the canals; that part of it in the pulp chamber was removed and the mummifier pressed into the chamber. Pulp involvement was caused by caries. It was extracted for reasons stated under Figure 1. The pericemental membrane is normal, with no evidence of root filling.

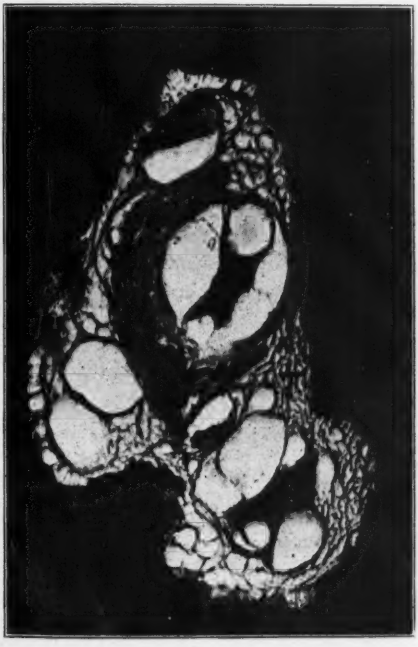


Fig. 11.—One millimeter from the apex of the lingual root. *T*, atrophic, and *H*, hypertrophic, tissue; *S*, secondary dentin; *X*, absorption. (From tooth shown in Figure 10.)

or it may subside after causing only an increase in the amount of blood to the part, with the resultant sensitiveness of the tooth for a few days. In either case, the normal reconstruction of the pulp stump has been interfered with. We must remember that we have a wound, caused by the severing of the pulp somewhere near the apex of the tooth. An increase in the amount of blood to the peri-

Fig. 12.—Upper second bicuspid of I. K., aged 19, retained four years after treatment by arsenic and eugenol. The pulp was removed because of involvement by caries. The root filling consisted of guttapercha points. It was extracted because of extensive further decay, which the patient did not wish repaired. The pericemental membrane is doubtful, being partly hidden by the line marking the floor of the antrum, with the canal filled to within 2 mm. of the apex, but with some space between the filling and the canal walls. A better roentgenogram would undoubtedly have shown apical involvement.

cemental membrane, due to irritation after the pulp proper has been removed, will result only in a congestion of blood in the wounded area, with an increase in the number of leukocytic cells. This produces a condition very favorable to the so-called "weeping of a wound," with the exudation of blood plasma into the root canal; and immediately we have furnished the best possible culture medium for any bacteria that may be present.

This brings us to the point of the complexity of the wound caused by pulp removal. Talbot,[5] Grove,[6] Callahan[7] and many other American investigators would determine an average of from 70 to 80 per cent of all teeth as having multiple apical ramifications. Hess,[8] in a thorough study of thousands of teeth, shows the frequency of accessory canals varying from about 25 per cent in central incisors to as high as 80 per cent in upper third molars, with an average for all teeth of about 45 per cent. These accessory canals play an important part in the tissue changes that are to follow pulp removal. They are, however, no more cause for worry than the irregularities in the main canals. The dentin partitions, the narrow channels connecting canals, the enlargements and constrictions of canals, frequently produce conditions almost as impossible of complete mechanical pulp removal as do the accessory canals. The tissue changes fol-

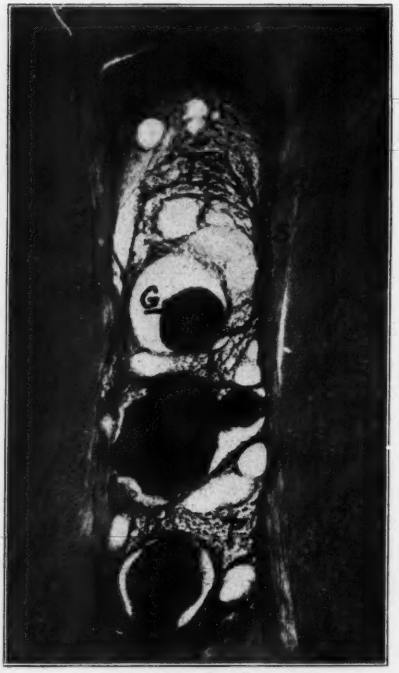


Fig. 13.—About 5 mm. from the apex. *G*, guttapercha points; *S*, secondary dentin; *T*, atrophic degenerating tissue surrounding points. Much leukocytic infiltration is present in this tissue. Similar tissue with leukocytic infiltration was found in the tissues at the apex. (Compare the roentgenray (Fig. 12) and histologic appearance of the root filling.)

Fig. 14.—Lower second molar of O. H., aged 57, retained three years after treatment by procain pressure anesthesia and formocresol. The pulp was removed because of hyperemia due to occlusal wear. The canal filling consisted of an antiseptic and guttapercha points. It was extracted for reasons stated under Figure 5. The pericemental membrane at the apex is thickened with one canal point extending to within 2 mm. of the apex, whereas the other canal has no filling evident.

5. Talbot, E. S.: Histopathology of the Apical Dental Tissues. Abnormal Collateral Arterial Development of the Roots of Teeth, Dent. Cosmos, 61:827 (Sept.) 1919.

6. Grove, C. J.: Biology of Multi-Canaliculated Roots, Dent. Cosmos, 58:728 (July) 1916; Mouth Infections, with Causes and Treatment for Periapical Abscesses, J. N. D. A., 4:1206 (Nov.) 1917.

7. Callahan, J. R.: Multiple Apical Foramina of the Roots, J. N. D. A., 3:85 (March) 1916.

8. Hess, Walter: Anatomy of Root Canals of the Teeth of the Permanent Dentition, New York: William Wood & Co., 1925, pp. 42-44.

lowing the severing of the pulp at all these places will depend materially on the condition of the pulp at the time of its removal, and on the method of treatment instituted.

In order that favorable tissue changes may take place, this treatment must be definitely aseptic, and the medicaments used must comply with biologic laws which will permit of the retention of vitality by the unremoved apical tissues. If this is done, the resistance of the accessory canal tissues seems equal to that of the apical tissues proper. In some cases studied, the degeneration of the tissues of the main canal foramen was very marked, while the tissue in the accessory canals took a better stain and seemed to have a more nearly normal vitality. (Figs. 15 and 18.) The formation of secondary dentin also seemed proportionally greater in these accessory canals, showing a greater vital reaction. (Fig. 6.)

Fig. 15.—Apex of root. *M*, mummified tissue; *C*, main canal, with some evidence of absorption; *A*, accessory canals all containing hypertrophic degenerating tissue, with much secondary dentin (*S*), and no evident absorption. There is some round cell infiltration in accessory canals and more in the pericemental membrane. The accessory canal tissues were not affected by the formocresol and remained vital. (From tooth shown in Figure 14.)

CALCIFICATION AND ABSORPTION

The conditions of calcification and absorption of the hard tissues in the cases studied were similar to those reported by previous investigators. Some cases show evidence of absorption followed by calcification (Fig. 9), which indicates a possible severe irritation at the time of pulp extirpation, causing absorption, and as the irritation lessened following root filling, the process of calcification began. In some, a second process

of absorption, following previous absorption and calcification, seems to be associated with a change in the general condition of the remaining pulp tissue, tending toward a breaking down of that tissue. (Fig. 11.) The two processes seem to be associated with varying degrees of irritation, the more severe causing absorption, which is of osteoclastic character.[9]

Fig. 16.—Lower first molar of patient O. H., aged 57, retained eight years after treatment by procain pressure anesthesia and formocresol. The pulp was removed because of hyperemia due to occlusal wear. The canals were filled with guttapercha points and eucapercha. It was extracted for reasons stated under Figure 5. There is pericemental thickening at the apex of both roots, with the canals filled in the mesial root to within 3 and 6 mm. of the apex, and in the distal root to within 4 mm. of the apex.

It is therefore contended that a tooth supported by a normal pericemental membrane is not to be condemned even if the root filling does not extend to the apex. If the pericemental membrane is normal, we can feel assured that the apical tissues are either normal or are undergoing a gradual reconstruction leading up to a stable condition. In all the specimens studied, where the roentgen ray showed a normal apical pericemental membrane, vital tissues were found in the canal foramen and extending some distance into the canal. In none of these cases was there any evidence of leukocytic infiltration that would indicate infection. As soon as we come into the class in which a definite thickening of the pericemental membrane is evident, we begin to find the presence of various forms of leukocytes, indicating the presence of infection. It is these teeth concerning which doubt is manifest as to the judgment that is to be passed. We have all seen vital teeth that have had a thickened apical pericemental membrane, possibly from traumatic occlusion. There may or there may not be infection in root-filled teeth having a thickened pericemental membrane, and all facts in the case concerning the patient's physical condition, based upon a thorough medi-

Fig. 17.—Tip of the mesial root. *T*, atrophic degenerating tissue and many accessory canals. There is no round cell infiltration. (From tooth shown in Figure 16.)

9. Skillen, W. G.: Hard Tissue Changes Noted Within the Canals of Treated Teeth and Their Possible Significance, J. A. D. A., 11: 350 (April) 1924.

cal examination, should be weighed and balanced before final judgment is passed on such teeth.

In teeth having large areas of apical involvement, all tissues in the canal proper were so nearly destroyed as to leave little for histologic description. Frequently, no tissue at all was found in the canals of these teeth, and where any was present, it usually appeared as a hyaline tissue with no evidence of cell outline.

Fig. 18.—Tip of distal root. *M*, mummified tissue in main canals and in many accessory canals. There is no round cell infiltration. The difference in the effect of the drug on the apical tissues of the two roots is probably due to accessibility of the distal canal.

COMPARISON OF HISTORIES AND HISTOLOGIC RESULTS

The condition found in each of the teeth studied can be interpreted only by referring to the roentgenograms of these teeth. The treatment used and the conditions found to exist do, in most cases, show a definite relationship. In some, strong medicaments used were prevented from affecting the apical structures by intervening unremoved pulp tissue, and the apical tissues remained vital. (Figs. 3 and 8.) In others, the pulp was thoroughly removed, and the effect of strong medicaments used was transmitted directly to the periapical structures, resulting in pericemental thickening or in the formation of an area of involvement. (Fig. 20.) In all of the cases studied, the use of an antiseptic drug after pulp removal seemed indicated. This should

Fig. 19.—Upper cuspid of G. F., aged 30. The pulp in this tooth died and the tooth was opened, Sept. 19, 1922. It was found to have no periapical involvement, and was treated with formocresol, September 19, 22 and 29. The canal was filled with guttapercha points, October 2, after which the tooth was retained for three years. It was extracted for reasons stated under Figure 1. The apical pericemental membrane is thickened on the distal surface of the root, with the canal filled to within 4 mm. of the apex.

Fig. 21.—Lower first molar of J. W., aged 27, retained five years after pulp removal by procain pressure anesthesia, and immediate canal filling with guttapercha points without further treatment. The guttapercha point extends through the apical foramen in the distal root, and there is a large area of involvement. The mesial root is filled to within 1 mm. of the apex and shows a thickening of the pericemental membrane.

Fig. 20.—Lateral termination of the main canal, with pericemental membrane attached. *M*, mummified tissue extending into the pericemental membrane as a result of treatment, with splitting of the pericemental membrane. There is much round cell infiltration. (From tooth shown in Figure 19.)

not be sufficiently strong to injure tissue with which it comes in contact, but should be strong enough to inhibit bacterial growth.[10] This view harmonizes with the general tendency at present to use drugs which will conform to the biologic principles involved, permitting the retention of vitality by the apical structures so that a normal reconstruction of those tissues may follow.

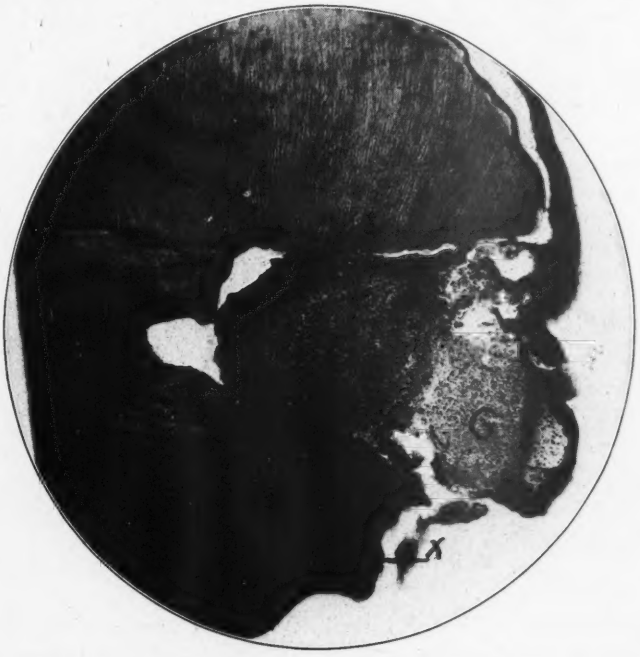


Fig. 22.—Tip of distal root. *G*, canal points extending through apical foramen; *E*, pericemental membrane with marked round cell infiltration; *S*, secondary dentin; *X*, absorption of root end.

DISCUSSION

F. C. Husband, Toronto, Canada: The slides shown by Dr. Moen have demonstrated in effective manner the difficulties of the root canal problem. We recognize that the vulnerable portion of the root canal is the apical third, owing to the multiple foramina. One of three things must be accomplished to render this area safe from infection. We must (1) mechanically obliterate these foramina, or, in other words, seal them; (2) keep them sterile to permit physiologic closure as suggested by Clyde Davis; (3) introduce into this portion of the canal a material that will permanently keep it sterile and at the same time be nonirritating to the soft tissues. I would like to ask Dr. Moen if he could suggest any other way to deal with this portion of the canal. Crownwise of this portion, we have little to fear, since the dentinocemental junction is impervious to micro-organisms, preventing their ingress or egress.

10. Coolidge, E. D.: Clinical Results in Root Canal Treatment, J. A. D. A., 14:493 (March) 1927.

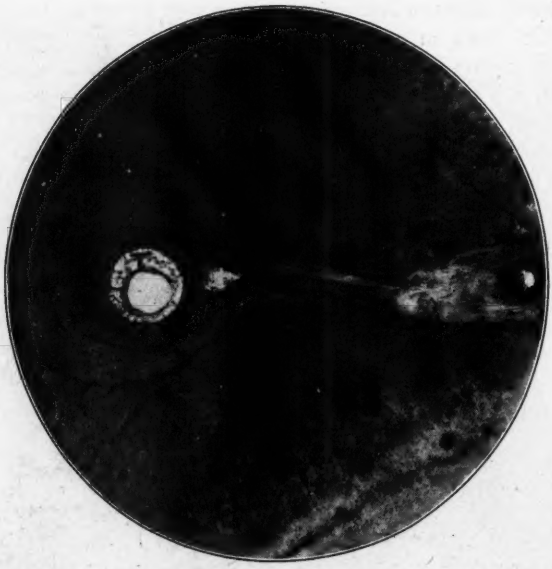


Fig. 23.—Tip of mesial root. *T*, atrophic degenerating tissue with no leukocytic infiltration; *S*, secondary dentin; *X*, absorption prior to laying down secondary dentin.

TISSUE CHANGES IN TRAUMATIC OCCLUSION*

By BALINT ORBAN, M.D., Chicago, Ill.

IN the following specimens, I shall demonstrate to you a few cases of "traumatic occlusion"[1] and show the tissue changes that may occur owing to this condition. The first case is that of a 24-year-old human being who died of tuberculosis. Figure 1 shows the right side of the upper and lower jaws in occlusion. In the upper jaw, the bicuspids are missing, and the first molar has a gold crown. The second molar has a deep cavity on its mesial-buccal side, and is broken down so that it is not in occlusion. The lower first molar is missing, and the second molar is tipped mesially. The lower second molar is in occlusion with its mesial cusp only. The second lower bicuspid on this side is not in occlusion.

*Read before the Section on Oral Pathology at the Midwinter Clinic of the Chicago Dental Society, Jan. 24, 1928.

*From the Research Department of the Chicago College of Dental Surgery, Dental Department of Loyola University.

1. The first who called attention to the significance of traumatic occlusion is M. Karolyi, Vienna: Oester-Ung. Vierteljahrsschr. f. Zahnheilk, 1902.

The left side of the same jaw is seen in Figure 2. The upper second bicuspid is missing; the first molar has a gold crown. The lower first molar is missing, and the second molar is tipped toward this space. The occlusion is somewhat different from that of the right side because the upper molar is in contact with the mesial cusp of the lower second molar and with the distal margin of the second bicuspid. In this type of occlusion, the lower second molars of both sides are tipped at every closure of the mouth toward the mesial side. The lower left second bicuspid is tipped somewhat distally, and the lower right second bicuspid

Fig. 1.—Right side of the jaw of a 24-year-old human being.

is not in occlusion at all. This kind of occlusion may be considered as "traumatic," and we shall see the tissue changes which occurred in this case. I possess the sections of the whole jaw of this individual, but for a simpler explanation, I shall describe only the changes apparent in the lower second molars and second biscuspids. The changes in the upper first molars are quite similar.

Figure 3 is a section through the lower left side (Fig. 2) showing the bicuspid and the second molar. Both are somewhat tipped toward the space left by the extracted first molar. The continuity of the alveolar bone around the teeth, especially around the bicuspid, is broken only by blood and lymph vessels. Some changes may be noted in the alveolar bone of the molar near the alveolar margin, but the low magnification shows no details.

The bone trabeculae of the jaw between the bicuspid and the molar are few and thin. The marrow is rich in cells. It is not the typical fatty marrow, but it contains many cells of the blood-building marrow. The marasmic condition of the tuberculous individual may account for this finding, and also for the

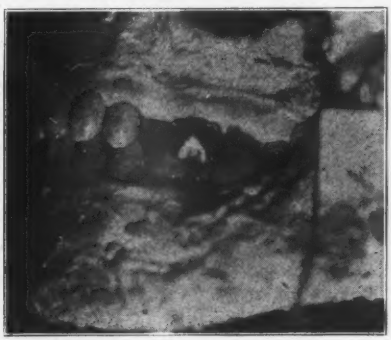


Fig. 2.—Left side of the jaw of a 24-year-old human being.

atrophic condition of the bone.

The picture of the right side shows no difference from that of the left in low magnification. The following photographs are higher magnifications of the periodontal membrane and surrounding tissues of both sides of the lower jaw. Figure 4 indicates the region where changes in the periodontal membrane of the second lower molars may be noted. Figure 5 shows the periodontal membrane of the molar of the right side, corresponding to the area *I* of Figure 4.

The alveolar bone (lamina dura) is not a continuous layer, but is interrupted in several places. The periodontal membrane at many places is in connection with the marrow spaces, the content of which is of a more fibrous type. In both marrow spaces and the periodontal membrane, wide blood vessels are numerous. The condition of the vessels and marrow spaces indicates a slight chronic inflammatory process, probably due to the proximity of the tooth to the bone. More serious injuries may be seen on the left molar (Fig. 6), deeper in the periodontal membrane toward the apex, corresponding to area *II*. The periodontal membrane is compressed. The whole is deeply stained, showing no differentiation between the fibers, nuclei and protoplasm. The entire periodontal membrane is necrotic. No resorption of the alveolar bone from the periodontal membrane has taken place, for there are no living cells which could resorb. The adjacent marrow space near the bone is fibrous; further away, it is very cellular. What is peculiar in this case is the formation of new bone trabeculae on the side nearest the marrow spaces, as seen in this photograph.[2] The surface of the bone is covered by a continuous layer of osteoblasts, which build new bone trabeculae, the arrangement of which recalls the so-called osteophytes. This type of bone structure is found in cases of chronic inflammation or irritation. Whether the development of this type of bone in this case is due to the traumatic irritation or to the

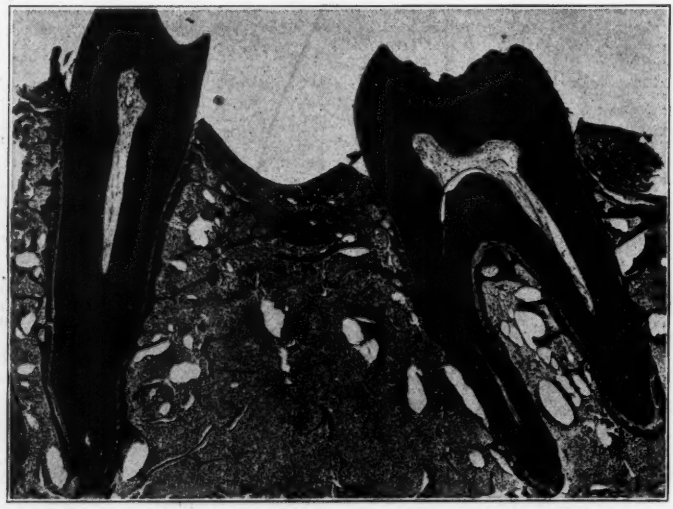


Fig. 3.—Section through the lower left second bicuspid and second molar. The molar is tipped mesially.

2. C. Haupl described similar findings: Ztscher. f. Stomatol., 25: 307, 1927. Haupl, C., and Lang, F. I.: Paradentitis Marginalis, 1927.

tuberculous condition or to both cannot be decided.

To return to our case, we observe changes near the margin (*I*) and in the middle (*II*) of the mesial periodontal membrane. The changes in the depth of the periodontal space have been more severe, and indicate that the force applied to the tooth (articulation only with the mesial cusp) has caused more damage in the depth than at the margin. This fact may show that the fulcrum of the tooth

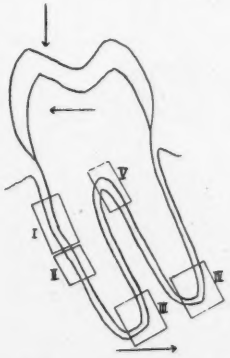


Fig. 4.—Five areas where injury on the periodontal membrane and surrounding tissue of the lower right and left molars can be observed. The arrows indicate the forces which occurred.

is not at the margin of the alveolus but, as has been supposed by C. S. Case, at the center of the root.[3] If we follow the periodontal membrane toward the apex, we find normal conditions on the mesial side, and find changes only at points *III-IV*, corresponding to the diagram.

Figure 7 shows both apices of the left molar, corresponding to *III* and *IV* in the diagram. The mesial periodontal membrane is normal, no resorption of bone being noted, and the regular principal fibers being present. On the distal side, hemorrhage, necrosis of the periodontal membrane, and resorption of bone can be seen. The same changes occur in the bifurcation, as may be observed in Figure 8. If the whole series of sections is followed, it can be stated that the greatest damage in the bifurcation is not exactly on the crest, but more toward the mesial side of the distal root, corresponding to *V* in the diagram. (Fig. 4.)

Before describing pictures of higher magnification, I again call attention to the localization of the changes in the periodontal membrane and surrounding bone. Changes may be noted at the mesial side on the alveolar margin (*I*) and center (*II*) of the alveolus, distally to the apices *III-IV*. In the bifurcation, changes may be noted more toward the mesial side of the distal root *V*. This localization of the injury shows exactly the nature of the trauma. The articulation between the upper first molar and the mesial cusp of the lower second molar has continually pushed the lower molar toward the mesial alveolar bone. As the tooth is a two-armed lever with the fulcrum near the center of the root, we find the damage mesially toward the middle and the marginal region, and at the apex distally, as shown in the diagram. Figure 9 shows an area of the periodontal membrane on the distal side of a root of the lower molar.[4] At one place, the periodontal membrane is missing. Between the surface of the cementum and bone, there is a space partly filled with débris, and darkly

3. Remarkable proof of this fact recently has been established by the experiments of A. M. Schwarz, Vienna: Ztschr. f. Stomatol., 26: 40. 1928.

4. The high magnifications do not always correspond to the lower magnifications but are different sections in the series.

stained. At this place, the tooth has been forced so close to the bone that the periodontal membrane has become necrotic, and cementum and alveolar bone lie in contact with each other. No resorption of bone has occurred at this place, owing to the absence of living connective tissue.[5] This bone, being in contact with the tooth, becomes resorbed from the side where the bone borders on the living periodontal membrane. This may be seen at the upper and lower end of the picture. New bone trabeculae, as osteophytes, are built from the side of the marrow space. The marrow is of a fibrous nature. A high magnification of one of the sections illustrating the process of resorption is shown in Figure 10. No resorption has occurred from the periodontal membrane where the tooth

Fig. 5.—Periodontal membrane of the lower right molar corresponding to area *1* in Figure 4. *a*, dentin; *b*, cementum; *c*, periodontal membrane; *d*, alveolar bone; *e*, marrow space; *f*, wide communication between periodontal membrane and marrow space.

5. Similar findings have been recently reported by Gottlieb on experimental traumatic occlusion: Fortschr. d. Zahnheilk., 1928, p. 398.

and alveolar bone are in contact; only a few necrotic pieces of the membrane remain at this point. From the side of this point, resorption of the bone has occurred

Fig. 6.—Periodontal membrane of the lower left molar corresponding to area *II* in Figure 4. *a*, dentin; *b*, cementum; *c*, cementum resorbed; *d*, necrotic periodontal membrane; *e*, new building of bone.

without corresponding resorption of the cementum. Newly built bone trabeculae may be seen on the side of the marrow spaces lined by osteoblasts. It is supposed that this type of bone formation is due to inflammation, which in this case may be caused by trauma.

Somewhat removed from the greatest injury, we see resorption taking place in some places on the surface of the teeth also. Figure 11 shows such an area. Large osteoblasts resorb the cementum and dentin. The periodontal membrane has lost its principal fibers and is composed of a loose, soft, connective tissue interwoven with many capillaries. This is a granulation tissue. In the upper end of the picture, we see the normal principal fibers of the periodontal membrane. At some places on the side of the bone, resorption may be seen; at others, new bone formation.

Similar findings as described are noted in connection with the lower second molars being in traumatic occlusion, and in the case of the upper first molars. But what is more remarkable, necrosis, resorption and new building of osteophytes like bone trabeculae are to be found also in the upper and lower incisors, but not to the same extent as in the lower second molars. This fact suggests that the main trouble in the case of this individual is not the trauma in the occlusion but the general condition due to tuberculosis and general marasmus. In studying this series of sections, the conviction was reached that if the condition producing this injury, i. e., the traumatic occlusion, could be overcome, the damage could not be repaired. There is no doubt that the tuberculosis of the individual is in marked degree responsible for the condition in the jaws. Only improvement of the general condition, with the correction in the occlusion, would help to repair the damage in the jaws. At several places in the periodontal membrane, as shown in Figure 12, there is resorption of the necrotic tissue by giant

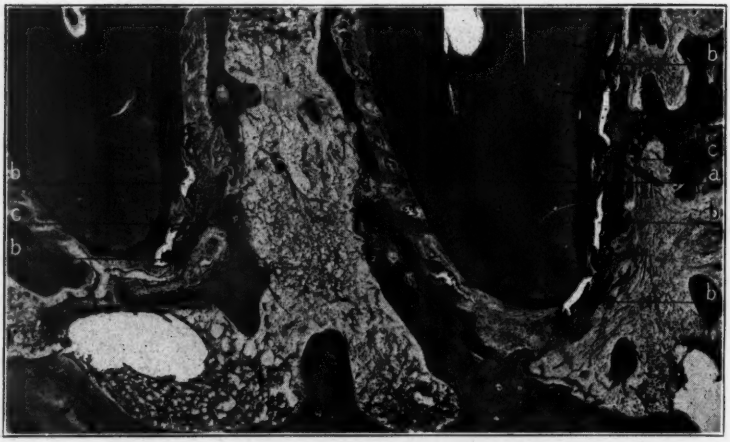


Fig. 7.—Both apices of the lower left molar corresponding to areas III and IV in Figure 4. *a*, normal periodontal membrane, on the mesial surface of the roots; *b*, necrosis of the periodontal membrane, with bleeding distally from the apex; *c*, new building of bone.

Fig. 8.—Bifurcation of the lower right molar corresponding to area *V* in Figure 4. *a*, dentin; b, cementum; *c*, periodontal membrane; *d*, interradicular septum; *e*, granulation tissue near the necrotic tissue; *f*, necrotic tissue; *g*, new building of bone.

cells. In such cases, after the necrotic pieces of bone and blood coagulum are resorbed or organized, we have every right to assume that new cementum may be deposited on the surface of the tooth, to the surrounding bone, by the building of new alveolar bone.

The following pictures will demonstrate the differences between the periodontal membrane of a functioning and

Fig. 9.—Periodontal membrane distally from the apex. *a*, **dentin;** *b*, **cementum;** *c*, **alveolar bone;** *d*, **necrotic tissue. The alveolar bone at this area lies in contact with the tooth. Between them, we find only necrotic tissue.** *e*, **resorption from the side parts where normal periodontal membrane is to be found;** *f*, **new building of bone trabeculae.**

and normal conditions may be restored. The periodontal membrane fibers again become attached to the tooth by means of the deposition of new cementum; and a nonfunctioning tooth. Figure 13 is the periodontal membrane of the lower left second bicuspid in articulation with the mesial cusp of the upper first molar.

The injuries that may be noted on the apex of this bicuspid are not so serious as the injuries on the molars. Normal periodontal membrane exists on the sides of the root. The principal fibers of the periodontal membrane run obliquely from the bone to the cementum. Their ends are embedded in each structure in which the fibers may be followed.

Figure 14 is the apical region of the lower right second bicuspid, which is not in occlusion. In this low magnification, it may be seen that the fibers of the periodontal membrane are not so regularly arranged as those originating from the left bicuspid. (Fig. 13.) The periodontal membrane is compressed and necrotic on the apex of the mesial side. There has been no occlusion at this tooth; yet there is definite injury. It is certain that during mastication this tooth was sometimes in function. Large pieces of food certainly became lodged between the upper molar and this bicuspid. Under normal conditions, this would have produced no injury, but, in this case, be-

Fig. 10.—*a*, dentin; *b*, cementum; *c*, necrotic tissue; *d*, alveolar bone which becomes resorbed from the side (*e*); *f*, granulation tissue; *g*, new bone trabeculae.

cause the principal fibers of the periodontal membrane are, for the greater part, missing, even this slight stress caused injury. A high magnification, Figure 15, shows clearly that the principal fibers in most places are missing, and that the fibers of the periodontal membrane are running parallel to the surface of the root. It is evident that a tooth which loses its principal fibers cannot resist even slight stress. The absence of fibers in this case is probably due to the loss of function caused by the missing antagonist.

The practical conclusion based on this case needs no words. Lost teeth must be replaced to keep the others in good condition.

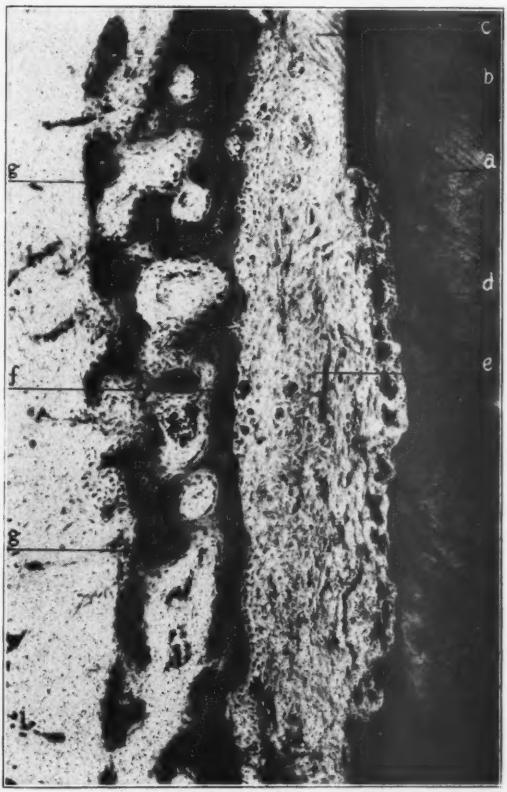


Fig. 11.—*a*, dentin; *b*, cementum; *c*, normal periodontal membrane; *d*, resorption on the surface of the tooth; *e*, granulation tissue between tooth and bone; *f*, resorption on the surface of the bone; *g*, new building of bone.

I call attention to the fact that, in Figure 14, no resorption is seen on the surface of the cementum in the nonfunctioning tooth, which has been supposed

to occur by some writers. Kellner has shown that the cementum of nonfunctioning teeth in several cases was thicker, and the periodontal membrane narrower than it was in teeth in function.[6] For an explanation of this, I refer you to the original paper. The comparison

local, but a question of the physical condition of the individual. Gottlieb first called attention to this fact in his paper on diffuse atrophy of the alveolar bone.[7] He demonstrated a case in which, as a result of trauma, new building of cementum occurred. Figure 16 is a specimen

Fig. 12.—Resorption of the necrotic periodontal membrane. *a*, dentin; *b*, cementum; *c*, blood and bone remaining are resorbed by osteoclasts; *d*, bone; *e*, resorption of bone.

of Figures 13 and 15 also verifies the findings of Kellner.

We have just seen in this case the damage done by traumatic occlusion, and have derived the lesson that the whole question of traumatic occlusion is not

of Gottlieb's work. Here are spikelike areas of cementum hyperplasia elongating in the direction of the fibers of the periodontal membrane. No destruction of any kind is evident in this specimen. It was taken from a 50-year-old human

6. Kellner, E.: Ztschr. f. Stomatol., 26: 271, 1928.

7. Gottlieb, Bernhard: Ztschr. f. Stomatol., 21: 195, 1923.

being. For a detailed description of the case and of Gottlieb's view, I refer you to his paper. Gottlieb's conclusion, based on his histologic findings, is that the resorption of alveolar bone and the destruction of the periodontal membrane in traumatic occlusion depend on the traumatic occlusion will lead to the development of cementum hyperplasia, and so to the strengthening of the teeth (Gottlieb's case). As Gottlieb pointed out, it is not possible that teeth which have been erupted and in good occlusion for from ten to twenty years could sud-

Fig. 13.—Periodontal membrane of the lower left second bicuspid. *a*, dentin; *b*, cementum; *c*, alveolar bone with embedded fibers; *d*, stretched principal fibers.

physical condition of the individual. Different individuals react in different ways to the same outer influences, as, in this case, occlusion. In some individuals, the normal occlusion will be traumatic (the front teeth of the tuberculous case previously described). In other cases the denly be in traumatic occlusion without any cause. Clinicians sometimes demonstrate traumatic occlusion thus: The patient closes the mouth, the two teeth, for instance two bicuspids, meet before the others, and the demonstrator claims they are in traumatic occlusion. They

say that the trauma is the cause of the loosening. As Gottlieb pointed out, this is misleading, for it is quite possible that a few weeks or days previously, the teeth were in normal occlusion. It is not possible that two teeth from 1 to 2 mm. longer than the others can exist in occlusion in a jaw for years. The relative increase in length is due to some more or less unknown primary cause. The traumatic occlusion appears as a secondary factor. It is evident that we must correct the occlusion by grinding down the teeth and must do everything possible to save the supporting structure from the damage caused by the secondary factor—traumatic occlusion. The damage done by this traumatic occlusion has been shown in the case described.

The following case will demonstrate also how much the general condition has to do with this problem. Figure 17 is the right side of the jaw of a human being about 40 or 50 years old. The occlusion was very bad. The lower bicuspids at the right side of the jaw, as well as the upper bicuspids, are ground down in different directions. As the

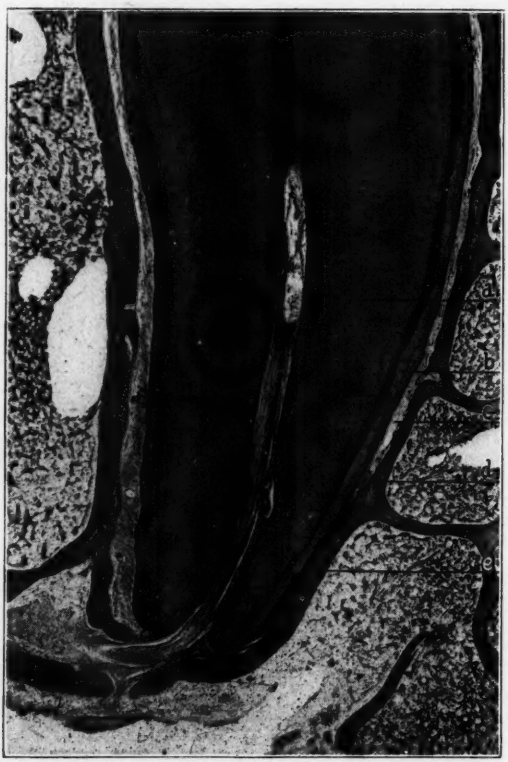


Fig. 14.—Apical region of the lower right biscuspid. *a*, dentin; *b*, cementum; *c*, periodontal membrane; *d*, alveolar bone; *e*, necrotic area at mesial portion of the apex.

second bicuspid is the last tooth in the lower jaw, the upper molars have erupted more than normally. From this condition of articulation, it is evident that the two bicuspids had hard work to do. A section through the two bicuspids is shown in Figure 18. We observe long roots and a normal, almost continuous, alveolar bone around the roots. The alveolar margin is high, almost reaching the cemento-enamel junction. The periodontal membrane shows ideal conditions, with normal principal fibers, as seen in Figure 19, a higher magnification which reveals the apical region. No resorption in bone or cementum, no nec-

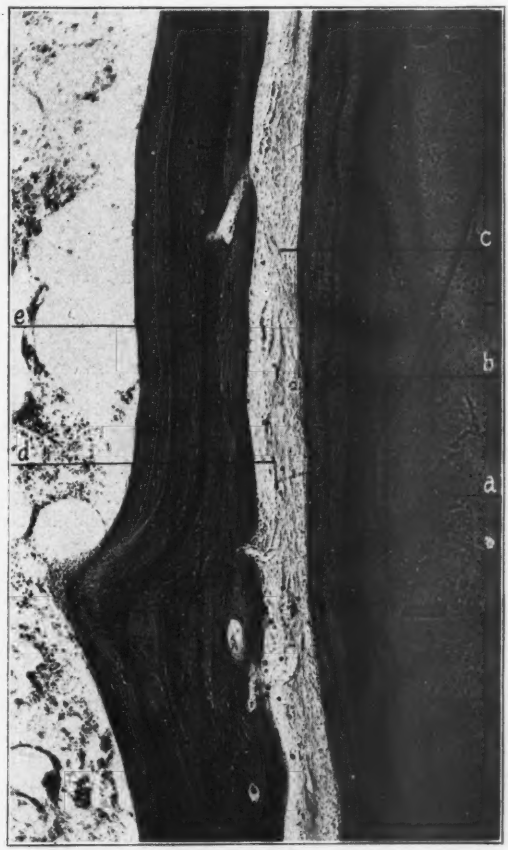


Fig. 15.—Higher magnification of Figure 14. *a*, dentin; *b*, cementum; *c*, a few stretched fibers in the periodontal membrane; *d*, periodontal membrane. In the greater part of the periodontal membrane (*d*), the fibers are running parallel to the surface of the tooth. *e*, alveolar bone; no embedded fibers.

Fig. 16.—Spikelike cementum hyperplasia on the root of a tooth exposed to extensive stress. *a*, dentin; *b*, spikelike cementum hyperplasia; *c*, alveolar bone; *d*, periodontal membrane.

Fig. 17.—Right side of a human jaw (40-50 years). Note the occlusion of the bicuspids.

Fig. 18.—Section through the lower bicuspids shown in Figure 17.

rosis, no granulation tissue nor inflammatory change can be seen.

No mention has been made until now of pyorrhea for the reason that pyorrhea means pus flow from the gingival crevice, which has little to do with traumatic occlusion. It may, in a neglected case, leading to secondary damage, be a predisposing factor in the development of a crevice and must always be corrected. Figure 20 is the gingival crevice at the mesial side of the lower right second molar of the first case shown. An almost normal epithelium covers the surface. Little sign of subepithelial inflammation and no ulcers are in the crevice epithelium. The inflammation of the outer epithelial tissue in this case is not more widespread than it is in any other case in which there is as much calculus as is evident here. There is no pus flow, no pyorrhea, in this case. Pus flow from a gingival crevice can never be cured by

merely correcting the occlusion, but it may often be cured by treating the crevice without treating the occlusion.

I shall not attempt to discuss this subject in detail, but I shall emphasize the fact that pyorrhea should be divided into two groups, as Gottlieb has divided it.

Fig. 19.—Apical region of the first bicuspid shown in Figures 17 and 18. *a*, dentin; *b*, cementum; *c*, alveolar bone; *d*, periodontal membrane normal all around.

One group is "pyorrhea," a more or less local condition of the gum. Gottlieb designates this state as "filthy pyorrhea," as it is due to an unhygienic condition of the mouth. The other group is "atrophy of the alveolar bone." In early stages, the two may be easily differentiated; later, they may be confused. The early symptoms of bone atrophy are wandering of the teeth and loosening of the teeth without crevice formation and without pyorrhea. Later, crevices and pyorrhea may develop.[9]

Weinmann, a pupil of Gottlieb, has found that patients with bone atrophy

9. It seems that Harold Keith Box accepted this classification of Gottlieb's in his recent work on the gingival crevice. Box describes exactly the same symptoms as does Gottlieb. We shall have more to say about this work of Box in another connection.

have a peculiar metabolic disturbance.[8] He found that patients in this condition have a normal basal metabolism, but have a low metabolism reaction to proteins. For instance, if a normal person eats from 100 to 150 gm. of protein, his metabolism will increase from about 20 to 40 per cent in the next hour or hour and a half. Individuals with bone atrophy show an increase under 20 per cent. The significance of this condition is not yet quite clear. Further investigation along this line may lead to a successful causal treatment accompanied by a symptomatic treatment, viz., treating the occlusion.

Fig. 20.—Gingival crevice and epithelial attachment on the mesial side of the lower right molar. *a*, dentin; *b*, enamel lost by decalcification; *c*, cemento-enamel junction; *d*, epithelial attachment; *e*, bottom of the gingival crevice; *f*, free margin of the gum; *g*, inflammatory infiltration; *h*, calculus. The space between the calculus and free margin of the gum is due to shrinkage during preparation.

8. Weinmann, J.: Ztschr. f. Stomatol., 25: 822, 1927.

TRIGEMINAL NEURALGIA AND ITS TREATMENT BY ALCOHOLIC INJECTION*

By ARTHUR E. SMITH, D.D.S., M.D., Los Angeles, Calif.

THE nervus trigeminus is the largest and most complicated of the twelve cranial nerves. The location of this nerve, with its principal branches almost entirely surrounded by bone, and its direct association with other nerves, makes it difficult to master and understand.[1] It is, no doubt, involved in more pathologic conditions, and subjected to more abuse, than any other nerve comprising the nervous system, owing to its location and its association with the contents of the orbit, sinuses, tonsils and teeth.

Its sensory fibers transmit stimuli of pressure, pain and temperature from the eye, nose, portions of the ear, mouth, the dura mater, anterior two-thirds of the tongue and the anterior portion of the scalp.

The two roots, a sensory and a motor, course anteriorly in the posterior fossa of the skull, entering the middle fossa and extending to the gasserian ganglion. (Fig. 1.) This large ganglion is located on the apex of the petrous portion of the temporal bone, and is connected with the ophthalmic, superior and inferior maxillary divisions.

The ophthalmic division makes its exit through the superior orbital fissure and supplies sensation to the contents of the orbit, part of the mucous membrane lining the nasal fossa, a portion of the skin of the forehead, the eyebrow and the nose. (Figs. 2 and 3.)

The maxillary division makes its exit from the skull through the foramen rotundum, crosses the sphenomaxillary fossa and enters the floor of the orbit, assuming the name infraorbital. In general, it supplies sensation to the nose, upper teeth, upper lip, lower eyelids, antrums, temple, cheek, forehead and soft palate. (Figs. 2 and 3.)

The inferior maxillary division passes from the skull through the foramen ovale. It enters the sphenomaxillary fossa, and it supplies sensation to the lower teeth, the alveolar process, the major part of the lower jaw bone, mucous membrane, periosteum, the skin covering the temple, external ear, lower lip, face and anterior two-thirds of the tongue. Its motor fibers supply motor impulses to the muscles of mastication, the anterior belly of the digastric muscle, and the mylohyoid muscles. (Figs. 2 and 3.)

THE DEEP ORIGIN OF THE NERVUS TRIGEMINUS

The nervus trigeminus arises from two roots: a large sensory root and a small motor root. The motor fibers arise

*Read before the Section on Oral Surgery, Exodontia and Anesthesia at the Sixty-Ninth Annual Session of the American Dental Association, Detroit, Mich., Oct. 27, 1927.

1. Smith, A. E.: Block Anesthesia and Allied Subjects, St. Louis: The C. V. Mosby Co., 1920.

from a nucleus in the pons, and from a column of cells throughout the midbrain. The sensory portion arises from large nerve cells located in the gasserian ganglion and ends in the sensory nucleus of the pons and medulla. (Figs. 2 and 4.)

TWO TYPES OF TRIGEMINAL PAIN

The trigeminus is subject to two types of neuralgia: (1) the symptomatic type, or atypical neuralgia; (2) the spasmodic paroxysmal type, or tic douloureux. Before the classification of trigeminal pain is discussed, referred pain will be considered.

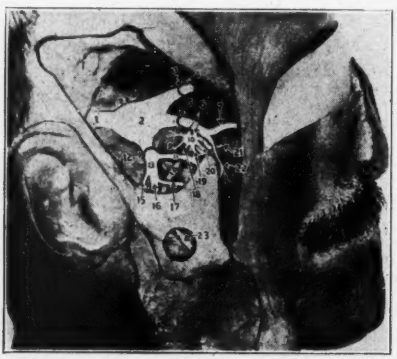


Fig. 1.—Author's wet anatomic specimen showing the following structure: *1,* sensory root of fifth nerve; *2,* gasserian ganglion; *3,* superior orbital fissure; *4,* ophthalmic division of fifth nerve; *5,* foramen rotundum; *6,* sphenopalatine nerves; *7,* infra-orbital nerve; *8,* vidian nerve; *9,* posterior palatine nerve; *10,* sphenopalatine ganglion; *11,* buccinator nerve; *12,* foramen ovale; *13,* mandibular nerve; *14,* internal maxillary artery; *15,* auriculotemporal nerve; *16,* inferior dental nerve; *17,* lingual nerve; *18,* middle palatine nerve; *19,* anterior palatine nerve; *20,* nasopalatine nerve; *21,* posterior lateral nasal nerve; *22,* posterior-superior dental nerve; *23,* inferior dental nerve.

REFERRED PAIN

Location of the etiologic condition causing referred pain may be difficult. Pain may be produced by either a distant or a local condition; for example, an irritated pulp may cause pain in its immediate region or may be located at a distant point, as in neuralgia in the ear or occipital headache. This phenomenon is due to the direct relationship of nerves. An important diagnostic feature, if the pain is referred, is the fact that the tissues in the region of interpretation are not sensitive to pressure; but, if the pain is not referred, the tissues in the area of interpretation are painful to pressure.

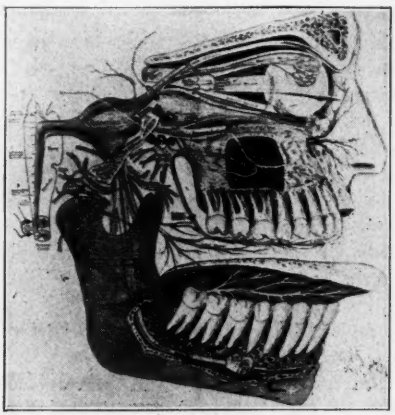


Fig. 2.—Nervus trigeminus and anatomic structures supplied by its branches. (Author's drawing.)

Referred pain may be caused by pressure, injury, trauma or irritation of a sensory nerve at a distant point. In such a condition, stimuli are transmitted over the sensory nerves to a remote part and interpreted as pain. A common example of referred pain, undoubtedly met by every dentist, is as follows: A patient complains of pain in a certain tooth. On examination, the tooth is found to be free from caries or irritation, and further examination reveals a carious cavity in the opposite jaw. It is, therefore, difficult to convince the patient that no dis-

ease exists in the tooth pointed out by him and that his pain is caused from a lesion in the opposite jaw.

In order to illustrate referred pain, I will use the sensory root of the fifth nerve, which is situated between the gasserian ganglion and its deep sensory nucleus. This large sensory nucleus is highly complicated and extends from the pons through the third cervical segment of the spinal cord. (Figs. 2 and 4.) At this point, the sensory branches of the nervus trigeminus are in close relation. Therefore, pain stimuli may arise, owing

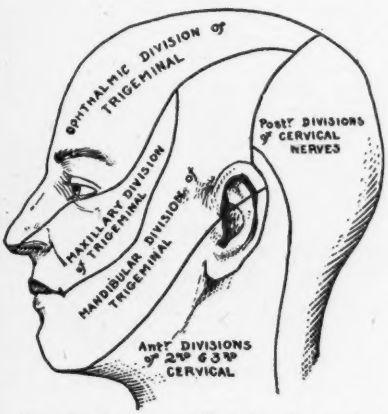

OPHTHALMIC DIVISION of TRIGEMINAL
Post^r DIVISIONS of CERVICAL NERVES
MAXILLARY DIVISION of TRIGEMINAL
MANDIBULAR DIVISION of TRIGEMINAL
Ant^r DIVISIONS 3d 2d 6 3d CERVICAL

Fig. 3.—Areas supplied by the various divisions of the fifth cranial nerve and cervical plexus. (Redrawn from Gray's Anatomy.)

to some pathologic condition in an accessory sinus, nasal cavity, eye, tooth or ear, and interpreted as pain at a distant point, such as the temple, occiput or other region, rather than the actual point of irritation.

Frequent pain stimuli, such as nasal, ocular, aural or dental, interpreted in the occipital region, are anatomically explained by the central contiguity of the ophthalmic, maxillary and mandibular afferent sensory terminals, to those of the upper three cervical nerve roots, located in the region of the descending trigeminal nucleus. It is a well known fact that a pathologic condition involving the tissues supplied by the nasociliary nerve may produce neuralgic pains in the forehead and scalp region. (Fig. 5.)

Other striking examples of referred pain are neuralgia in the ear or a frontal headache caused by the presence of a carious, impacted or malposed tooth, infection of the antrums or frontal sinus, pulp nodules, alveolar abscess or eye strain.

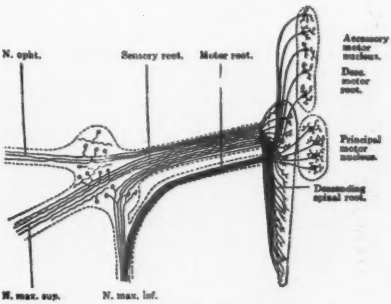


Fig. 4.—Nuclei of deep origin of the fifth cranial nerve. (From Howell's Physiology.)

There is no question that antral, frontal sinus and ocular disease, alveolar abscesses or the presence of decayed, infected, impacted, malposed or unerupted teeth, necrosed bone and dental cysts have played an important rôle in the symptomatic or atypical type of neuralgia, which may predispose to true trigeminal neuralgia or tic douloureux.

Malaria, focal infection from teeth and tonsils, syphilitic gumma, trauma, neuritis or some form of intracranial or cortical pathologic process, such as tumors, may be causative factors. The various forms of psychoneuroses, which may attack the fifth cranial nerve, are purely symptomatic, being secondary

manifestations of other pathologic lesions.

In order to give the patient relief, many nerve trunks have been needlessly blocked with alcohol. The operator did not locate and remove the causative factor, which can only be accomplished by a thorough examination of the patient.

ETIOLOGY OF SYMPTOMATIC PAIN OR ATYPICAL NEURALGIA

Atypical neuralgia is caused by pathologic lesions which yield to a different treatment from that of true trigeminal neuralgia or tic douloureux. The superior and inferior maxillary divisions are more susceptible to the so-called rheumatic influences, while the ophthalmic division is more susceptible to septic poisoning. In most cases, symptomatic pain, caused from the foregoing condition, will be arrested with proper surgical or medical treatment.

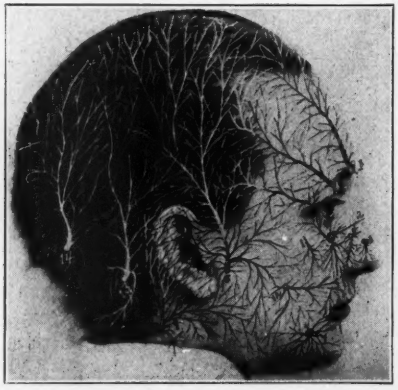


Fig. 5.—Sensory nerve supply of the face, forehead and scalp. *1*, mental nerve; *2*, infraorbital nerve; *3* and *4*, external nasal branches; *5*, auriculotemporal; *6*, zygomaticotemporal nerve from second division; *7*, supraorbital nerve from ophthalmic division; *8*, supratrochlear from ophthalmic division; *9* and *10*, greater auricular nerve; *11*, malar nerve; *12*, long buccal nerve; *13*, lesser occipital nerve; *14*, greater occipital nerve. (From author's textbook.)

ETIOLOGY OF TRUE TRIGEMINAL NEURALGIA OR TIC DOULOUREUX

The specific etiology is unknown. Microscopic examination of sections of the gasserian ganglion and nerve branches has revealed nothing definite. It has been my observation, in treating these cases, that arteriosclerosis is frequently present, the patient giving a history of some infection, together with that of the symptomatic or atypical neuralgic pains of the face. From these many observations, I am of the opinion that true trigeminal neuralgia, or tic douloureux, is caused by some toxic process, bringing about degenerative change in the gasserian ganglion. It appears to be a special disorder dependent

Fig. 6.—Insertion of the needle for blocking the second division of the fifth nerve. The needle is advanced upward, backward and inward. When at a depth of 15 mm., the needle point should strike the posterior lateral surface of the tuberosity, and is then advanced an additional 15 mm. along the periosteum. The total depth is approximately 30 mm.

on some pathologic change in the nerve itself.

In some cases, neurotic changes have been observed, such as deposition of connective tissue and increased vascularity, but the cause of this may be the result of former treatment, such as injection of alcohol. There may be impingement on the gasserian ganglion or nerve branch by a tumor, an exostosis or cicatricial tissue.

Horsley[2] was of the opinion that the disease is an ascending neuritis caused from dental caries, but conclusive proof has never been established. The symptoms of tic douloureux do not simulate those of neuritis. Frazier[3] is of the opinion that it is due to sclerosis of the gasserian ganglion. Dana has advanced the theory that it is caused by a degenerative process of the gasserian ganglion. Patrick[4] states that the pathologic changes which have been found do not afford sufficient proof to justify making a definite statement regarding its etiology.

Others have advanced the theory that it is due to an inflammatory condition of the ganglion and, in order to support their contention, state that the symptoms of tic douloureux followed an attack of epidemic encephalitis. However, this contention was not proved, because microscopic examination of sections of the gasserian ganglion failed to disclose any histologic change.

2. Horsley, Victor: Surgical Treatment of Trigeminal Neuralgia, Practitioner, 65: 251, 1900.

3. Frazier, C. H.: Am. J. Sc., 169: 469 (April) 1925.

4. Patrick, H. T.: Symptomatology of Trifacial Neuralgia, J. A. M. A., 62: 1519, 1914.

5. Adson, A. W.: Trifacial Neuralgia and Its Treatment, Northwest Med., 22: 156 (May) 1923.

Adson[5] has observed that tic douloureux is frequently associated with general sclerosis or cerebro-arteriosclerosis, but less frequently with multiple sclerosis, together with other neuralgic pains, i. e., atypical neuralgic pains involving the face and sciatic and intercostal neuralgic pains. In view of these observations, Adson states that he is convinced that the etiology of tic douloureux is similar to the etiology of the above named diseases; and since the pain is not relieved by removal of the foci of infection, it seems logical to assume that the lesion at the time that the neuralgic pain develops is not acute, even though one suspects it to be due to infection, and must, therefore, be due to some sclerotic lesion which has developed within the gasserian ganglion, representing a post-infectious result.

SYMPTOMATIC PAIN IN ATYPICAL NEURALGIA

As atypical neuralgic pains must be differentiated from those of tic douloureux, a complete history, by thorough examination and close observance of the patient, must be obtained. Conclusive points in making a diagnosis are (1) the duration of the disease, and (2) the duration and character of the pain. If the neuralgic pain is caused from infection of the antrum, frontal sinus, teeth or tonsils, the presence of impacted, unerupted or malposed teeth, pulp stones, pus infections, malaria, tumors or any other acute condition, it will, if observed over sufficient time, reveal characteristics which will differentiate it from true trigeminal neuralgia or tic douloureux. If the pain is caused by any of the foregoing conditions, it generally remains as a continuous dull boring ache. The pain attacks the patient slowly. At the onset, the pain is mild, the degree and intensity increasing in accordance with its cause

and the progress of the pathologic derangement. If caused by an impacted lower third molar or pulp stone, the onset would be slow and less severe than if it is caused by some acute infection. The pain of symptomatic neuralgia may be increased in intensity when the patient lies down. It may prevent sleep and is more or less constant. Symptomatic neuralgic pain is not paroxysmal in type, being diffuse in character, and may extend beyond the median line or involve tissue not supplied by the trigeminal nerve.

SYMPTOMS OF TRUE TRIGEMINAL NEURALGIA OR TIC DOULOUREUX

The symptoms of tic douloureux are characterized by agonizing shooting pains, usually starting in the nasal region, cheeks or lips, and radiating through the teeth and jaws to the orbit, forehead, temple, ear and base of brain. In the beginning, the individual may attribute the pain to toothache, and he will seek relief from his dentist. If dental disease is discovered and checked, the dentist and patient may have the opinion that the causative factor has been removed. However, after a lapse of days, weeks or months, the patient will experience a second attack, usually of greater severity than the initial attack and before securing dental service.

Each attack may last from a second or two, with a twinge of sharp lancinating pain, to a period of two minutes of pain, which is agonizing, lancinating, sharp, shooting and characterized by a burning sensation.

Paresthesia of the affected side of the face and areas called "trigger zones" are present. A "trigger zone" is a hypersensitive circumscribed area of skin, usually about 2 cm. in diameter, which may, or may not, be located in the skin supplied by the affected nerve. In mandibular nerve involvement, it is commonly located just laterally to the ala of the nose. In rare cases, it may be located on the opposite side of the face. Increased lacrimation, discharge from the nose, spasmodic contraction of the facial muscles and tongue may occur, with the patient groaning, slapping and rubbing the face and manifesting an expression of extreme agony.

DIFFERENTIAL DIAGNOSIS

Successful treatment depends upon a correct diagnosis. Symptomatic pain or atypical neuralgia must be differentiated from true trigeminal neuralgia or tic douloureux.

One of the most important diagnostic aids in arriving at a diagnosis is roentgenograms of the teeth, osseous structures, antrums and accessory sinuses. Transillumination is of some value. A Wassermann test should be made to rule out syphilis. In most cases, the onset of the symptomatic or atypical neuralgia is gradual, this depending, however, on the nature of its cause. If it results from an alveolar abscess, frontal or maxillary sinus infection, the onset will be more rapid, the pain more severe, than if caused from a tumor, pulp stones, foci of infection located in the teeth or tonsils or a cyst or sclerosed bone following tooth extraction. Symptomatic or atypical neuralgic pain may be caused from toxic absorption, neuritis, irritation of the terminal nerve endings or pressure on a sensory nerve. If caused from an acute condition, some evidence of inflammation may be observed. Palpation over the affected area usually reveals tenderness, but no "trigger zone." If caused from some dormant condition, there will be no signs of inflammation and an absence of tenderness to pressure. As a rule, the pain is diffuse and it may radiate across the median line and extend to the neck

and shoulders. It is generally augmented on retiring and causing loss of sleep. The pain is generally constant. Remissions may occur, but the periods between attacks are different from those of tic douloureux.

Clinically, true trifacial neuralgia may simulate atypical neuralgia in some of its symptoms, and it may prove difficult to establish a diagnosis. In tic douloureux, the pain appears in cycles, with periods of remission. It is confined to tissues supplied by the fifth nerve and does not cross the median line, and, at the beginning, the patient may take little notice of it, thinking it may result from a carious tooth, or that he has taken cold, giving it no further thought until a second attack occurs. At this time, the patients generally seek relief from physician or dentist. They may have carious teeth filled and pulpless teeth, or even all the teeth and the tonsils, removed. Such operations, if the patient is afflicted with true trigeminal neuralgia, will prove of no avail. Quite true, restorative dental operations, removal of infected teeth and tonsils and the draining of a sinus may apparently seem to be the causative factor, and the completion of such operations, together with the apparent cessation of painful paroxysms is, indeed, a happy coincident. But in the not distant future, the patient experiences another attack.

Usually, at first, the pain is localized and without a "trigger zone," but as the disease progresses, paresthesia and "trigger zones" develop. Primarily, the pain may be located in one branch of the fifth nerve, but later a second branch, or all three branches, may become involved. A paroxysm may be elicited by taking food, moving the tongue, brushing the teeth, drinking from a glass, touching the finger to the face or feeling a draft of cold air or by the slamming of a door. In taking food or drink, the paroxysm is more likely to be elicited if the substance taken is either sweet or sour, but the fact that the food is hot or cold is not a factor in bringing about the paroxysm. Therefore, it should not be confused with pulpitis.

Usually, the pain returns in one definite spot, radiates to a certain location and involves only the side of the starting point. Invariably, during a paroxysm, the patient suddenly slaps his hand heavily over the affected part and begins a vigorous massage, all the while smacking the lips and drawing the mouth to the opposite side. In severe cases, the patient may refrain from taking proper nourishment for fear of another attack. Depression of spirits is present in many cases, because of intense agony, no doubt the severest pain Nature could devise. The patient experiences more difficulty during the winter, and may escape an attack during the summer months.

I have observed only one patient with bilateral tic douloureux. This condition is not associated with headache; therefore, the history of periodic attacks of radiating pain to the orbit, and over the forehead, such as are present in migraine, rules out true trigeminal neuralgia.

Sphenopalatine neuralgia, as described by Sluder,[6] is a pain at the root of the nose, about the eye and upper jaw, extending beneath the zygoma to the ear, and taking on the form of earache. The pain is severest distally from the mastoid, extending backward to the occiput and neck, and may extend to shoulder and arm. The patient may describe the pain as an "aching" in the throat and pain inside the nose. The pain is not so severe

6. Sluder, Greenfield: Headaches and Eye Disorders of Nasal Origin, St. Louis: The C. V. Mosby Co., 1918.

as that of tic douloureux, and is not paroxysmal. Sphenopalatine neuralgia is ruled out by blocking the sphenopalatine ganglion with procain.

Glossopharyngeal neuralgia is located in the region of the glossopharyngeal nerve, and the "trigger zone" is in the tonsillar region. The pain is intermittent and paroxysmal, and if it is caused from involvement of the glossopharyngeal nerve, anesthetizing the throat will block the "trigger zone." A paroxysm, if one can then be elicited from the face, indicates trigeminal nerve involvement and tends to rule out glossopharyngeal neuralgia.

TREATMENT OF TIC DOULOUREUX

Treatment is three-fold in character: (1) block anesthesia; (2) nerve avulsion, and (3) severance of the sensory root of the gasserian ganglion.

Only the block method of treatment will be discussed in this paper. Other methods will be mentioned briefly.

There is no specific drug treatment for the cure of tic douloureux. Matas says:

> A vast therapeutic and pharmacological arsenal has been brought into play for the relief of this most painful and distressing affection. While there is no specific drug treatment for the cure of the confirmed type of facial neuralgia, a knowledge of the resources of pharmacological therapeutics is indispensable for the symptomatic relief of pain. Innumerable and most ingenious methods of neurectasis and neurectomy have been devised for the cure of this condition. Some of these are still of value for the relief and possible cure of the peripheral forms in certain classes of subjects who are physically unfit to undergo any trauma. In practice, at the present time, the treatment of the essential and confirmed disease of the trigeminus is limited to a choice of: (a) the alcoholization of the nerve trunks at their origin from the basal foramina; (b) alcoholization of the gasserian ganglion; (c) intracranial division or avulsion of the sensory root of the fifth nerve posterior to the ganglion.

NERVE AVULSION

Nerve avulsion is practiced with only a limited degree of success. The nerve branches which have been resected are: infraorbital, supraorbital, nasal and inferior dental. Temporary relief may follow, but invariably pain of a more severe type returns. When considering the limited relief given patients, it appears to be indicated only in individuals who cannot undergo the major operation for severance of the sensory root or who have become immune to the effects of alcoholic injections.

Fig. 7.—Correct position of needle and extension hub for blocking second division. A 10 mm. length of the needle is located exteriorly to the puncture and approximately 30 mm. within the tissues.

SEVERANCE OF THE SENSORY ROOT OF THE FIFTH NERVE

During the past few years, surgery for the treatment of tic douloureux has made rapid developmental strides. Gasserectomy, with its high mortality rate, has been replaced by the severance of the sensory root posterior to the gasserian ganglion.

With the modern operation, hemorrhage is controlled, the motor nerve is preserved and trauma is lessened, the results being in striking contrast to the shock, hemorrhage, motor paralysis, trauma and high mortality following gasserectomy. However, the modern operation for the severance of the sensory root is by no means void of mortality and complications, such as numbness of the face and a secondary trophic lesion of a neuroparalytic character of the eye. Modern development in the technic of this major operation has, in a great measure, been due to the efforts of Frazier,[7] Spiller, Van Gehuchten, Cushing,[8] Kanavel,[9] Adson,[10] Dowman, Coughlin[11] and Davis.

Fig. 8.—First position of the needle for blocking the second division of the fifth nerve by the intraoral route. The point of puncture is located in reflection of the mucous membrane situated between the cheek and gum tissue, buccally to the upper third molar.

TREATMENT BY ALCOHOLIC INJECTION

Blocking the nerves in the treatment of tic douloureux has been practiced since 1903, when Schlosser and Matas described the method of injecting alcohol. Since then, the technic for blocking the individual branches and the gasserian ganglion has been simplified. Others who have contributed liberally in its development are Oswald, Sigard, Levy, Harris,[12] Patrick,[13] Offerhaus, Hartel, Martin and Allen.

The ideal treatment effecting a cure of tic douloureux should be free from postoperative complications. Two methods of treatment, namely, the blocking with alcohol and the severance of the sensory root, have their indications and contraindications.

The intent of this article is to discuss the injection of alcohol, with the results obtained from its use when skillful technic is employed. Some surgeons strongly

7. Frazier, C. H.: Surgeon's Impression of Trigeminal Neuralgia Based Upon Experience with 302 Cases, J. A. M. A., 70: 1345 (May 11) 1918; Clinics N. America, 5: 1521 (Dec.) 1925.

8. Cushing, Harvey: Surgical Aspects of Major Neuralgia of the Trigeminal Nerve. A Report of Twenty Cases of Operation on the Gasserian Ganglion, with Anatomic and Physiologic Notes on the Consequences of Its Removal, J. A. M. A., 44: 773, 1905.

9. Kanavel, A. B., and Davis, L. E.: Surgical Anatomy of the Trigeminal Nerve, Surg. Gynec. Obst., 34: 357 (March) 1922.

10. Adson, A. W.: Diagnosis and Surgical Treatment of Trigeminal Neuralgia, Ann. Otol. Rhin. Laryn., 35:601 (Sept.) 1926.

11. Coughlin, W. T.: Section of Sensory Root of the Fifth Cranial Nerve Under Local Anesthesia, Surg. Gynec. Obst., 33: 424 (Oct.) 1921.

12. Harris, Wilfred. Neuritis and Neuralgia, New York; Oxford University Press, 1926.

13. Patrick, H. T.: Treatment of Trifacial Neuralgia by Means of Deep Injections of Alcohol, J. A. M. A., 49: 1567, 1907.

advocate the major operation, placing the alcoholic nerve block in the same category with the time honored palliative drugs. In perusing the literature and observing the wide variation of results obtained from deep alcoholic block, conclusive proof is found that faulty technic has no doubt played an important rôle in securing mediocre results. The deep blocking of the superior and inferior maxillary nerve trunks and the gasserian ganglion is a surgical procedure, demanding aseptic technic, thorough knowledge of anatomy and skill in making the injection.[14] Matas says, "The alcoholization of the ganglion is a surgical procedure which requires experience, judgment and knowledge of technic quite equal to that of the surgical extirpation of the ganglion."

EFFECT OF ALCOHOL ON NERVE TISSUE

Alcohol has a destructive action on nerve tissue. Skin or muscle is not affected to the extent of loose connective or nervous tissue. It has the greatest destructive action on highly organized tissue, such as nerve trunks, ganglions and brain tissue.

The tissue degeneration is that of aseptic necrosis. Afferent stimuli cannot reach the brain, owing to interruption or severance of nerve fibers and destruction of ganglion nerve cells. The degenerated nerve tissue is partly replaced by connective tissue. Repeated alcoholic injections increase the deposition of connective tissue.

The successful outcome of a deep nerve alcoholic block injection depends on several factors, among which are: (1) the quantity and percentage of alcohol injected; (2) the injection of the alcohol directly into the nerve trunk of the ganglion; (3) the amount of nerve destruction caused by the alcohol, and (4) strict anatomic technic.

If the injection has been improperly executed, nerve destruction will be inadequate, resulting in rapid nerve regeneration, with the return of symptoms of tic douloureux. Greatly in favor of the alcoholic treatment is the fact that if the symptoms return, the injection can be repeated.

Fig. 9.—Wet specimen illustrating the position of the needle and extension hub for blocking the second division of the fifth nerve. The needle is started in the reflection of mucous membrane buccally to the upper third molar and the extension hub is situated between the occlusal planes of the teeth.

Many hold the view that there are various cases in which relief following alcoholic injections seems to be permanent. Nevertheless, if it is not permanent, it should be chosen as the method of election in all cases prior to the major operation. Harris says, "That if the nerve

14. Smith, A. E.: Local Anesthesia, J. N. D. A., 8: 749 (Sept.) 1921.

has been properly blocked, immunity can be guaranteed for twelve months, and four and five years is quite a common period."

Dalziel[15] says, "Injections of alcohol into the nerves will give relief from pain for a period varying from one to ten years, or even more."

Blair's observations have been that alcoholic injections give relief for a longer time than does peripheral nerve avulsion.

prevented infiltration of the nerve. He also reports two cases coming under his observation in which the patients had been relieved for twelve years.

Bauer,[16] of Stockholm, recently reported alcoholic injection treatment in twenty-four cases from 1915 to 1924. A total of fifty-two injections were made. The neuralgia was from two months' to twelve years' standing; the average age was 56. He states that thirteen of the twenty-four patients have so far been

Fig. 10.—Needle located laterally to the tuberosity and third molar, also anterolaterally to the posterosuperior dental nerve and foramen. *1*, posterior superior dental foramen; *2*, posterior superior dental nerve; *3*, second division of the fifth nerve crossing sphenomaxillary fossa.

Adsen reports, in a series of 839 cases, 2,338 alcohol injections, which, when successful, afforded relief for from nine to eighteen months on the average. Approximately 30 per cent of the injections were incomplete because of scar tissue; or the nerve could not be located. This was caused by previous injections and

cured, six greatly improved. One committed suicide; one showed no improvement, and in three, no postinjection results were reported.

Four hundred and forty-two cases of trigeminal neuralgia have come under my observation, covering a period of twelve years. This series of cases includes the symptomatic or atypical and

15. Dalziel, George: Method of Alcohol Injection in Trigeminal Neuralgia, Glasgow M. J., 106:15 (July) 1926.

16. Bauer, G.: Hygiea, Stockholm, July, 1926, p. 513.

the true trigeminal neuralgia or tic douloureux types. The total number of cases will be grouped under two headings:

1. *The Symptomatic or Atypical Neuralgia Type.*—From the total number of 442 afflicted with trifacial pain, 286 presented the symptomatic type, and the removal of the pathologic cause gave relief. The etiologic factors are classified as follows: (a) lesions of terminal nerve endings caused from burns, injuries, pulp stones or infections from teeth, sinuses or tonsils; (b) some pathologic process involving the main branches of the fifth nerve, such as infectious processes of the tissues, impacted or unerupted teeth, fractures of fibrous union, cysts, tumors and broken off hypodermic needles, and (c) systemic conditions resulting from septic poisonings, syphilis, etc.

2. *True Trigeminal Neuralgia or Tic Douloureux Type.*—Diagnosis was made in 156 cases. The etiologic factor could not be determined. The location of pain was as follows: (a) first or ophthalmic division, 1; (b) second or superior maxillary division, 25; (c) third or mandibular division, 37; (d) ophthalmic and superior maxillary divisions, 9; (e) superior maxillary and mandibular divisions, 63; (f) ophthalmic, superior maxillary and mandibular divisions, 21.

There were eighty-seven men and sixty-nine women, with an age range of 23 to 82; average, 55. Eleven were under 40, and 145 past 40. The right side

Fig. 11.—Wet specimen illustrating the needle in correct position along the posterior lateral portion of the tuberosity for blocking the superior maxillary division of the fifth nerve. *1*, long buccal nerve; *2*, lingual nerve; *3*, inferior dental nerve; *4*, mandibular nerve; *5*, auriculotemporal nerve; *6*, foramen ovale; *7*, posterior superior dental nerve; *8*, foramen rotundum; upper *9*, second division of the fifth nerve crossing the sphenomaxillary fossa; lower *9*, posterior superior dental foramen.

Fig. 12.—Posterior lateral view of the needle for blocking the second division of the fifth nerve by the intraoral route. The needle is inclined mesially. Approximately 15 mm. of the needle is in contact with the periosteum just below the second division. *1*, posterior superior dental foramen; *2*, second division of the fifth nerve.

was affected in 104, the left side in fifty-one and both sides in one.

Of the 156 cases diagnosed as tic douloureux, 293 deep block injections were given. The operation for peripheral nerve avulsion was performed on twenty-nine, with the following results: no relief in six; partial relief in eight; relief in twelve, the average being eleven months. Results were unknown in three cases. Seventeen of the 156 patients underwent the major operation for the severance of the sensory root.

Relief was given from deep alcohol block, as follows: in seven, from 10 to 11 years; fourteen, 6 to 9 years; twenty-nine, 3 to 5 years; sixty-one, 1½ to 2 years; twenty-nine, 6 months to 1 year; two, no results; fourteen, postinjection results unknown.

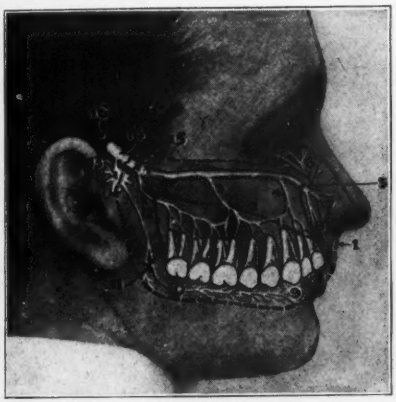


Fig. 13.—Area anesthetized by a second division injection which is indicated by the outline marked *1*. The solution is injected at the point marked *X*, indicated at *15*. *2*, nasopalatine nerve; *3*, middle superior dental nerve; *4*, anterior superior dental nerve; *5*, infraorbital foramen and nerve; *6*, infrapalpebral nerve; *7*, lateral nasal nerve; *8*, superior labial nerve; *9*, overlapping of nasopalatine and anterior palatine nerves; *10*, anterior palatine nerve; *11*, posterior palatine foramen; *12*, posterior superior dental nerve; *13*, sphenopalatine ganglion; *14*, second division of the fifth nerve; *16*, foramen rotundum.

Fig. 14.—Palpating the lower border of the zygoma, condyle, coronoid process and upper border of the ascending ramus for locating the sigmoid notch.

BLOCKING THE SUPERIOR MAXILLARY DIVISION OF THE FIFTH CRANIAL NERVE BY THE INTRAORAL METHOD

There are three technics employed for blocking the superior maxillary division of the fifth cranial nerve trunk, i. e., intraoral, extraoral and orbital. Only the intraoral technic will be discussed.

In 1913, I began to experiment with a method for blocking this nerve intraorally.[17] The results attained have been highly satisfactory. In fact, the extraoral route is used only in case of intraoral infection. The technic of injection was established after numerous injections of methylene blue and dissections on cadavers.

17. Smith, A. E.: Nerve Blocking, Oral Health, 8: 439 (Nov.), 481 (Dec.) 1918.

TECHNIC OF INJECTION

An iridioplatinum needle, 40 mm. long, 22 gage, mounted in a curved extension hub, is employed. The patient is placed in a horizontal position. The oral cavity is sprayed with an antiseptic solution. The mucous membrane, gums and teeth in the region of the maxillary tuberosity are dried with gauze and warm air and the area is isolated with sterile gauze packs. Tincture of iodin is applied to an area 3 cm. in diameter.

Fig. 15.—Sigmoid notch. The straight line represents the lower border of the zygomatic process. The curved line indicates the upper border of the ramus. The cross indicates the starting point for the needle.

The syringe contains 2 c.c. of 2 per cent procain-epinephrin-Ringer solution. The patient's mouth should be half open, permitting the operator to extend the cheek laterally with the first and second fingers to expose the area for the needle. (Fig. 6.) The needle should enter the tissue at the highest point in the mucous fold, buccally to the apices of the upper third molar. (Figs. 6 and 8.) Care should be taken not to insert the needle near the gum tissue, as such a procedure prevents the needle from advancing correctly.

The needle is advanced upward, inward and slightly backward, into the mucous membrane, allowing the bevel to strike the periosteum covering the posterolateral surface of tuberosity at an approximate depth of 15 mm. After the needle reaches the posterolateral surface of the tuberosity, it is advanced in close contact with the periosteum for 15 mm., making a total depth of approximately 30 mm. (Figs. 10, 11 and 12.)

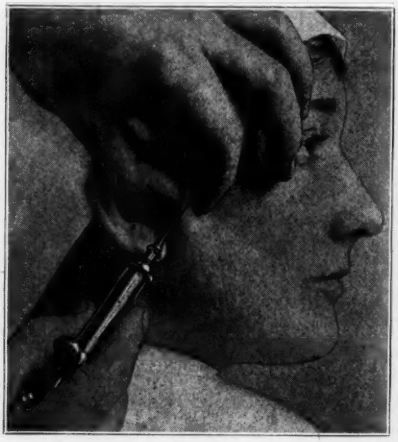


Fig. 16.—Preliminary injection into the skin prior to inserting the needle for blocking the third or mandibular division.

The extension hub should assume a position midway between the occlusal planes of the upper and lower teeth. (Figs. 7 and 8.) The distance from the puncture point to the second division of the fifth nerve as it crosses the sphenomaxillary fossa is from 27 to 33 mm. The needle should never assume a per-

pendicular course as, in such a position, it would not follow the tuber maxillae. (Fig. 12.)

If the needle is directed too far posteriorly, it will strike the inferior-lateral portion of the greater wing of the sphenoid or lateral portion of the external pterygoid plate. This indicates that the extension hub was held too high during insertion of the needle. The needle should be partly withdrawn and its direction changed. The route of the needle over the posterior-lateral tuberosity of the superior maxillary bone is void of

Fig. 17.—Needle in position while injecting the solution for blocking the third or mandibular division of the fifth nerve.

large blood vessels, and is located anterolaterally to the posterior superior alveolar nerve and artery as they enter their foramen. (Figs. 11 and 12.) The needle is from one-half to three-quarters inch anterior to the internal maxillary artery. (Fig. 1, *14*.)

INJECTION OF SOLUTION

One c.c. of a 2 per cent procain-epinephrin-Ringer solution is slowly injected. This procain injection has a two-fold purpose: (1) to make sure the needle point is near the nerve trunk, (2) to allay the pain caused by the deposition of alcohol.

If the procain has been deposited in contact with the nerve, signs of approaching anesthesia will be apparent within one minute, which indicates the contact of nerve and needle. A tingling sensation, indicative of approaching anesthesia, appears in the upper lip, lower eyelid and ala of the nose. The syringe is now detached from the needle (with the needle left in position), and is filled with 1 c.c.

Fig. 18.—Needle inserted through the sigmoid notch and in the region of the mandibular nerve; extraoral route.

of 90 per cent grain alcohol. The syringe is reattached (care being taken not to change the location of the needle), and the alcohol is slowly injected. (Figs. 7 and 9.) This injection blocks the superior maxillary division with all its branches. This method offers the shortest route to the nerve; the technic is more easily executed, with results more certain than by the extraoral or orbital

routes, and is easily and quickly carried out, without pain. (Fig. 13.)

BLOCKING THE THIRD OR MANDIBULAR DIVISION OF THE FIFTH CRANIAL NERVE BY EXTRAORAL METHODS

The third or mandibular division of the fifth cranial nerve is the largest of the three divisions, assuming the shape of the foramen ovale and varying in width from 7 to 12 mm. (Figs. 1 and 2.) After leaving the cranial cavity, it enters the infratemporal fossa, passes forward to an approximate distance of 1.5 cm. and then assumes an inferior-lateral course. (Fig. 2.)

Fig. 19.—Needle point 1 cm. anteriorly and inferiorly to the foramen ovale for blocking the third or mandibular division. *1*, foramen ovale; *2*, posterior part of the nasal septum.

Two needles are employed to make this injection. A fine (27 gage) needle is used for the preliminary injection. For the deep injection, a 20 gage, 60 mm. length is employed. The sigmoid notch is located with the index finger by palpating the condyle, the upper border of the ramus and the lower border of the zygoma. (Fig. 14.) With a dermographic pencil or iodin, a line is drawn along the lower border of the zygomatic arch, extending from the condyle to the coronoid process of the mandible, and another to outline the upper border of the ramus. These two lines should form a semicircle. (Fig. 15.)

The skin is surgically prepared and 1 c.c. of a 2 per cent procain-epinephrin-Ringer solution is injected beneath the skin at the point indicated by a cross in Figures 15 and 16. After surface anesthesia is secured, the 60 mm. needle is inserted at the point indicated by the cross, as shown in Figure 15, and is advanced directly inward at right angles to the surface of the skin. (Figs. 17 and 18.) The needle pierces connective tissue and masseter muscle.

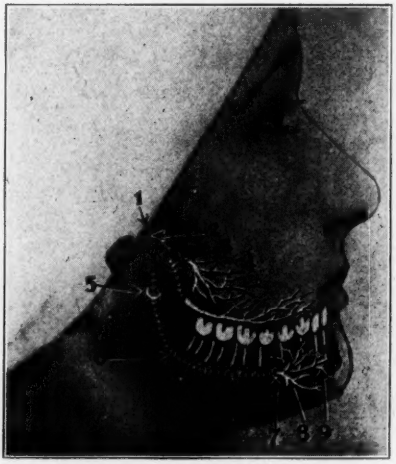


Fig. 20.—Outline of mandible and nerve supply. *1*, foramen ovale; *2*, mandibular nerve; *3*, lingual nerve; *4*, long buccal nerve; *5*, inferior dental foramen; *6*, inferior dental nerve; *7*, mental foramen; *8*, mental nerves and, *9*, incisive nerves.

Emphasis is placed on the necessity of advancing the needle into the tissues at right angles to the surface, as error is less liable to occur than in advancing the needle at a certain angle. The manibular division is reached approximately 5 mm. antero-inferiorly to the foramen ovale. (Fig. 19.) The approximate depth of the needle is 43 mm. Similar technic is now employed for the depth injection of procain and alcohol as above given for the superior maxillary nerve. Anesthesia is secured in the lower jaw, teeth and adjacent tissues. (Fig. 20.)

BLOCKING THE GASSERIAN GANGLION

Blocking the gasserian ganglion is strictly a surgical procedure, requiring experience, judgment and thorough anatomic knowledge. An attempt to block the gasserian ganglion or the deep nerve trunks without a thorough knowledge of the technic in all its phases will result in failure and the patient will be the victim of experimentation. Harris,[12] of London, was among the first to report a technic for direct injection of the gasserian ganglion. In 1914, Hartel described a technic of reaching the ganglion by the needle traversing the cheek without entering the oral cavity. (Fig. 21.) The technic of Schlosser, Oswald and Offerhaus, prior to Hartel's method, was somewhat objectionable because the needle reached the ganglion through the mouth, and there was a possibility of resultant infection. Hartel's method has been followed with success by many operators. He made an anatomic study of the relation of the ganglion to other structures, and pointed out several difficulties which might be encountered, especially those involving the internal carotid artery and cavernous sinus which are located internally to the ganglion.

With the Hartel method, the needle enters the cheek midway between the upper second molar and malar bone, and extends along a line midway between the occipital and parietal fontanels in the midline. A 10 cm. needle is directed upward, inward and backward between the ascending ramus of the lower jaw and the maxillary tuberosity. The needle is advanced in this direction until the smooth plate of the infratemporal surface is reached, which is located just anterior to the foramen ovale. The depth of needle by the Hartel method is approximately 6 cm. (Figs. 21 and 22.)

Hertzler[18] advises the insertion of the needle using a technic locating the parietal eminence with the thumb and the external occipital protuberance with the second finger, while the index finger marks a point midway between the two named landmarks. When the needle is forced in this direction, and should the foramen ovale be missed, it is usually found that the needle is too far mesially, which necessitates the retraction of the needle, which is advanced again more laterally.

Patrick[19] modified the technic as advocated by Baudouin, and Levy. Matas, Allen, Braun[20] and Offerhaus have contributed liberally to the advancement of alcohol blocking.

SMITH-NEIL TECHNIC FOR LOCATING THE FORAMEN OVALE

Dr. Ewell Neil and I[21] have endeavored to simplify the method of locating the foramen ovale and the technic for blocking the gasserian ganglion, and the clinical results obtained over a period of

18. Hertzler: Surgical Operations with Local Anesthesia, New York: Surgery Publishing Co., 1916.

19. Patrick, H. T.: Trifacial Neuralgia and Its Treatment, J. A. M. A., 64: 691, 1915.

20. Braun: Local Anesthesia, Philadelphia; Lea and Febiger, 1914.

21. Footnote 1, p. 810.

eight years have been highly satisfactory. Several hundred skulls were examined and many cadavers injected with methylene blue and then dissected, careful measurements being taken with a view to utilizing certain landmarks and developing a technic whereby the foramen could be located and the needle directed into the ganglion.

Locating the foramen ovale is the keynote which leads to successful blocking of the gasserian ganglion. By the Hartel route, the distance between the puncture

Fig. 21.—Injecting solution into gasserian ganglion by the Hartel route.

point, lateral to the angle of the mouth (as shown in Figures 21 and 22) and the foramen ovale is from 6 to 7 cm. This depth makes it very difficult to locate the foramen, and failure to do so necessitates the withdrawal of the needle, a change in its direction and readvancement.

During our experiments, it was found from adult skulls that the average distance between the external articular tubercle on the right and left sides was 128 mm. The length of the linea intertubercularis was ascertained by using special calipers graduated in millimeters. (Fig. 23.) The external articular tubercles are situated just anterior to the condyles, and inferiorly-externally to the zygoma. They are located by palpation, and the skin directly over them is marked with a pencil. Calipers are now carefully placed directly over the tubercles (Fig. 23), and, with slight pressure upon the skin, the reading is taken which indicates the linea intertubercularis from skin surface to skin surface. The average dis-

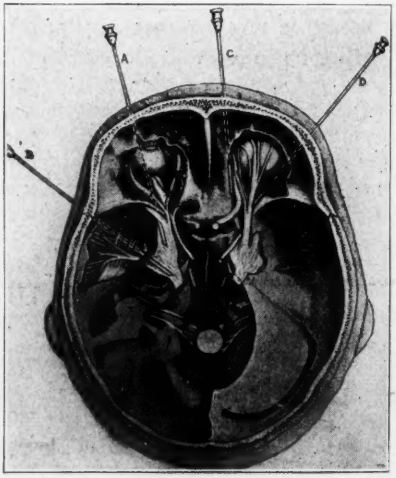


Fig. 22.—*A*, position of needle for blocking the gasserian ganglion through the foramen rotundum by the orbital route. *B*, position of the needle for blocking the gasserian ganglion through the foramen ovale by the Hartel method. *C* and *D*, positions of needles for blocking the ophthalmic division of the fifth nerve by Braun's method. (Redrawn from Hertzler.)

tance from the external surface of the tubercle to the center of the foramen ovale is nineteen-sixty-fourths of the total distance from tubercle to tubercle.

The average thickness of the skin, subcutaneous and muscle tissues, external to

the tubercle, is 5 mm., which is ascertained by anesthetizing the tissues with a local anesthetic, inserting the needle at right angles to the skin surface and advancing it until the tubercle is reached. (Fig. 28.) The tissues on both sides being 10 mm., that amount is subtracted from the skin to skin distance as obtained by the calipers. The following example, using the average measurements, will demonstrate the technic.

The total distance between skin surfaces, directly over the tubercles, we will say, is 138 mm. The thickness of the soft tissues on both sides is 10 mm. This, deducted from the total distance, leaves 128 mm., which is the length of the linea intertubercularis. Nineteen-sixty-fourths of 128 is 38 mm., which is the distance from the external surface of the tubercle to the center of the foramen ovale.

Fig. 23.—Author's graduated calipers in position over the external articular tubercles on the right and left sides.

THE USE OF THE TRIANGLE IN BLOCKING THE GASSERIAN GANGLION

This instrument was devised to direct the needle to the foramen ovale or into the gasserian ganglion. (Figs. 24 and 25.) The needle marked 6, Figure 30, graduated and 10 cm. long, is advanced into the tissues through guide 13. The guide is set in accordance to the depth of the foramen ovale. Solid needle 11 is passed through nut 8, the required distance. Guide 13 and needle 6 are set to form the angle, which is accomplished by placing the points of the needles together. This gives the angle at which needle 6 is inserted. (Figs. 24, 25 and 30.)

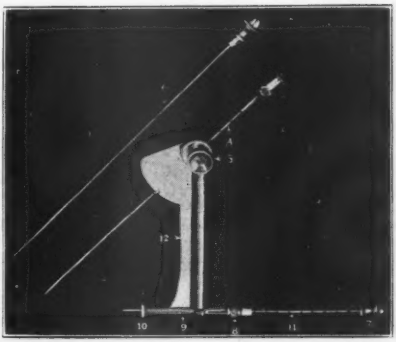


Fig. 24.—Triangle for blocking the gasserian ganglion. *A*, graduated needle; *5*, nut for holding guide; *6*, stylet for needle *A*; *7*, thumbpiece on solid needle; *8*, nut for locking needle *11*; *9*, guide for needle *11*; *10*, part which rests upon surface of skin; *11*, solid graduated needle; *12*, perpendicular of triangle.

The operator is aware of the location of the needle point and advances it the required depth through the foramen ovale into the ganglion. (Fig. 32.) This technic has been verified by injecting methylene blue into the ganglion of cadavers, following this by the exposure

of the ganglion by dissection. If the needle marked 11, in Figure 27, is advanced into the tissues just beneath the tubercle (Figs. 28 and 30*B*) at right angles to the skin surface until the point is resting over the center of the foramen ovale (Fig. 30*A*), and a perpendicular line is erected, allowing it to pass alongside the cheek (Fig. 27, *12*), opposite the buccal surfaces of the upper molar teeth until a depth of 9 cm. is reached, a base and perpendicular of a triangle are

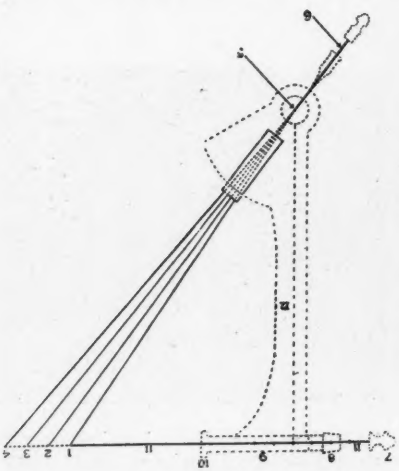


Fig. 25.—Triangle for blocking gasserian ganglion. *A*, graduated needle through which solution is injected; *6*, stylet in canula; needles *A* and *11* are perpendicular of triangle *12*, forming triangle.

formed. The hypothenuse of the triangle will pass through the cheek opposite the upper first molar between the coronoid process and the tuberosity of the maxilla (Fig. 28, *6*) into the foramen ovale at an approximate depth of 7 cm. The base of the triangle (Fig. 27, *11*) is capable of being adjusted to form various angles, and the hypothenuse is adjustable to form any angle necessary, and is easily adjusted for any fleshy or thin patient. Figure 26 shows the various angles which can be formed. Figure 27, *10*, is resting upon the skin when solid needle 11 is inserted under the tubercle of the zygoma and advanced into the tissues. Each needle is graduated in millimeters. After the distance of the linea intertubercularis is ascertained, the indicator, shown in Fig. 27, *5*, is adjusted to form the proper hypothenuse to complete the triangle.

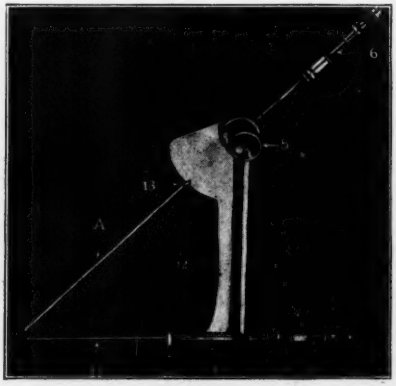


Fig. 26.—Principle of the triangle employed in the blocking of the gasserian ganglion. *1*, *2*, *3* and *4* represent various depths from the skin surface situated over the external articular tubercle at which depths the foramen ovale may be located. *5* to *1*; *5* to *2*; *5* to *3* and *5* to *4* are the various hypothenuses necessary to complete the triangle with the bases *9* to *1*; *9* to *2*; *9* to *3* and *9* to *4*. *6*, stylet and needle; *7*, thumbpiece on solid needle; *8*, lock nut; *9*, guide for solid needle; *10*, flattened portion of guide which rests on the skin; *11*, solid needle; *12*, perpendicular of triangle.

TECHNIC OF INJECTION

Locate the external articular tubercles and adjust the calipers. (Figure 23.) As an example, 138 mm. will be used as the skin to skin distance. Prepare the area for insertion of the needle by applying

alcohol and tincture of iodin. Make a superficial procain injection over the external articular tubercle. Insert a small needle at right angles to the surface, directly over the tubercle, to ascertain the thickness of the soft tissue. Assuming the depth to be 5 mm., adding a like depth on opposite side makes 10 mm. This, taken from 138, leaves 128 mm., the distance of the linea intertubercularis. Nineteen-sixty-fourths of 128 is 38 mm., representing the distance from tubercle to foramen ovale. To this, add the soft tissue measurement on one side, which is

Fig. 27.—Appliance in position as needles are advanced into the tissues. *5*, lock nut to hold guide for needle *6*; *8*, lock nut to hold solid needle *11*, after it is passed through guide *9*; *10*, flattened portion of guide resting over and below the external articular tubercle.

5 mm., giving a total of 43 mm., representing the distance from skin surface to center of the foramen ovale. Solid needle 11 (Fig. 26) is passed through guide 9, to a distance of 43 mm. (after it has passed the point marked 10) and is locked in position by nut 8, and needle 6 is adjusted at the proper angle. It must be remembered that 43 mm. is used here only as an example, and is the average distance needle 11 is inserted to reach the foramen. Needle 6 is adjusted to meet the point of needle 11; nut 5 is locked to hold the guide for needle 6. Nut 8 is loosened, and needles 6 and 11 are withdrawn.

The patient should be in a horizontal position. The skin is surgically prepared. Partial anesthesia is induced with nitrous oxid-oxygen for deep insertion of the needles. Two preliminary procain injections are made beneath the skin, one directly over the articular tubercle, the other 3 cm. laterally to the corner of

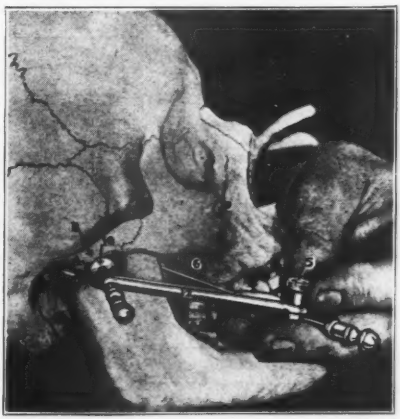


Fig. 28.—Lateral view of skull with triangle in position. *5*, lock nut to hold guide for needle *6*; *10*, flattened portion of guide resting anteriorly to and below the external articular tubercle, indicated at *A*; *12*, perpendicular of triangle; *B*, zygoma.

the mouth which is opposite the upper first molar. After waiting five minutes for surface anesthesia, adjust part 10 externally and below the tubercle (Figures 27 and 28), insert needle 11, 43 mm. and lock with nut 8 (Fig. 29). The point of needle 11 should be just outside the foramen ovale (Figs. 30 and 32) if the needle has been advanced at right

angles to the skin surface. When the mandibular nerve is reached, the patient will experience radiating sensations in the lower jaw.

The perpendicular of the triangle is held alongside the cheek of the patient until the end of the guide touches the skin opposite the upper first molar, approximately 3 cm. laterally and slightly above the corner of the mouth. (Figs. 27 and 31.) Direct needle 6 through guide 13 (Fig. 30), which has been previously set, advancing it through the tissues toward the foramen, the distance

Fig. 29.—Triangle in position while injecting the solution into the gasserian ganglion. *5*, guide for needle; *8*, lock nut which is holding the solid needle *11* in the guide *9*; *10*, flattened portion of guide resting over and below the external articular tubercle.

of which has been previously calculated and is easily ascertained by observing the graduations of the needle. The operator is thus aware when the two needle points have completed the triangle just outside the foramen ovale.

After this is accomplished, advance needle 5, 7 mm. through the foramen (never to exceed 1 cm.) into the anterior-inferior portion of the gasserian ganglion. As needle 6 is advanced (Fig. 27), it should be rotated to avoid deviation, often caused by the bevel of the needle. Remove stylet, attach syringe, and slowly inject 1 c.c. procain, epinephrin and Ringer solution, and observe results, especially as to whether or not the ophthalmic division is affected. (Fig. 29.) This injection accomplishes two things: (1) the rapidity of the tingling sensation of approaching anesthesia determines the location of the point of the needle; and (2) the anesthesia prevents

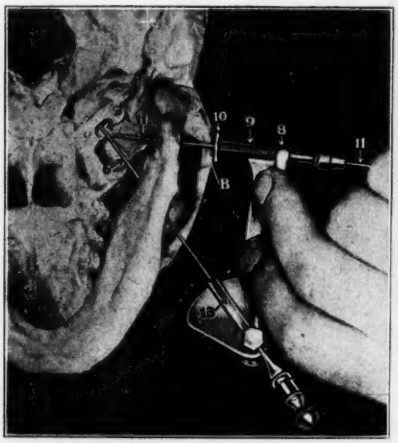


Fig. 30.—Inferior view of skull showing the triangle in position. *A*, foramen ovale; *B*, external articular tubercle of zygoma; *6*, needle and stylet inserted through the foramen ovale; *8* and *9*, guide for solid needle; *10*, flattened portion of guide; *11*, solid needle which forms angle with needle *6*; *13*, guide through which needle *6* is inserted.

pain from the alcohol injected. Symptoms of anesthesia should appear immediately in the superior maxillary and mandibular divisions.

Signs of anesthesia appearing primarily in the ophthalmic division indicate that the needle has entered the ganglion too

far and it should be withdrawn in accordance with the rapidity of approaching ophthalmic anesthesia.

Reattach the syringe and slowly inject 1 c.c. of 90 per cent alcohol for blocking the lower portion of the ganglion, the superior maxillary and mandibular divisions. Harris,[12] of London, advises that the alcohol be injected just interior to the lips of the foramen ovale. I prefer to insert the needle through the foramen into the lower portion of the ganglion. (Figs. 30 and 32.)

Fig. 31.—Triangle and needles in position on wet anatomic specimen for blocking the gasserian ganglion.

Fig. 32.—Triangle and needles in correct position for blocking the gasserian ganglion. *1*, sensory root of ganglion; *2*, gasserian ganglion; *3*, ophthalmic division; *4*, foramen rotundum; *5*, superior maxillary division; *6*, gasserian ganglion, with needle inserted through the foramen ovale into the ganglion; *7*, infraorbital nerve; *8* and *9*, posterior superior dental nerve and foramen; *10*, internal maxillary artery; *11*, solid needle, the point of which is located at the foramen ovale and forms angle with needle *6*; *12*, sphenomaxillary artery; *13*, long buccal nerve; *14*, foramen ovale; *15*, mandibular division; *16*, auriculotemporal nerve; *17*, inferior dental nerve; *18*, lingual nerve; *19*, coronoid process of mandible.

EFFECTS FOLLOWING ALCOHOLIC INJECTION OF THE GASSERIAN GANGLION

The usual signs of anesthesia are present after the procain injection. After the injection of alcohol, if the procain anesthesia is not pronounced, certain phenomena present themselves, as fulness or slight burning pain in the region of the vertex and jaws. After the procain anesthesia has passed, numbness prevails in the tissues supplied by the nerves blocked. Paresthesia and numbness remain from two to four weeks, then gradually disappear. The tactile sensation slowly returns, but there remains a loss of pain sense. Weakness of the muscles of mastication is manifested on account of the motor fibers being blocked. Motor paralysis of these muscles on the side of injection may be complete or partial, and may interfere with the patient masticating on the side blocked. As a rule, the patient will quickly learn to masticate on the unaffected side, and should be instructed against injury to the mucous membrane and the blocked side. The patient soon accustoms himself to the numbness. When the alcohol is in-

jected into the ganglion itself (and not at the foramen ovale), the motor nerve, being situated below, may not receive the full effect from the alcohol, this resulting in only partial incapacitation to the motor fibers. The motor sense or motor impulses return much more quickly than the sensory impulses and, in many cases, motor paralysis is hardly noticeable by the patient a few weeks after the injection. If too great a quantity of alcohol is used or it has been injected too rapidly, vertigo, dizziness, nausea or nystagmus may appear, or the pupil may become dilated and paralysis of the sixth cranial nerve may result. Deafness and facial paresis indicates that branches of the facial and auditory nerves have been affected by the alcohol. The degree of anesthesia of the ophthalmic nerve and numbness of the cornea depend on the depth of needle, the quantity of alcohol injected and the rapidity of injection.

If the alcohol is injected in the lower portion of the gasserian ganglion, the ophthalmic division, as a rule, is only partially affected; which is accounted for in two ways: (1) the small quantity (1 c.c.) of alcohol injected; (2) the fact that the outer and lower two-thirds of the ganglion (at which point the alcohol is injected) is composed of sensory fibers of the superior maxillary and mandibular divisions, while the inner one-third is composed of fibers comprising the ophthalmic division. (Fig. 4.)

Frequently, the ophthalmic branch is only minutely affected. However, partial anesthesia causes little postoperative concern. If anesthesia of the cornea results, the eye must be protected. With sensation impaired, or entirely absent, foreign material may enter and infect it. Keratitis must be guarded against. If the cornea becomes anesthetized, the eyelid should be covered with a Buller's shield, which is removed daily, the eye being irrigated with 2 per cent boric acid solution. The corneal shield and glasses, as advocated by Adson,[22] give gratifying results.

SUMMARY

Tic douloureux, or true trigeminal neuralgia, is a disease, the etiology of which is unknown. Without treatment, the paroxysms become more frequent and increase in their intensity and severity, making life almost unbearable, undermining the general health and incapacitating the individual from daily work.

Nerve avulsion of the peripheral branches gives only temporary relief, and then only in some cases. More satisfactory results are obtained from the alcoholic injection. Peripheral nerve avulsion is only indicated on those individuals who cannot undergo the major operation for the severance of the sensory root of the fifth nerve or who have become immune to the effects of alcohol injections.

Deep alcohol injections into the gasserian ganglion, the superior maxillary and mandibular divisions, should be given before subjecting the patient to the major operation, for two reasons: 1. If the injection has been properly given, the patient may be relieved of all pain for months, years or possibly life. The injections are not dangerous when properly executed, and can be repeated as often as necessary. 2. After a patient has undergone blocking with alcohol once or several times, he is better prepared to undergo the radical operation for severing the sensory root, as he has experienced the anesthesia and paralysis from the effects of the alcohol, which also fol-

22. Adson, A. W., and Schroeder, A. A.: A Corneal Shield, J. A. M. A., 86: 1835 (June 12) 1926.

low the major operation, and even to a more pronounced degree.

If the patient is suffering from true trigeminal neuralgia or tic douloureux, the removal of teeth, the draining of sinuses, the removal of cysts, pulp stones, etc., will prove of no avail. If the pain subsides after such operations, the operator was dealing with atypical neuralgia of the symptomatic type and not that of tic douloureux.

The radical operation should not be performed until the patient has been given the benefit derived from alcoholic injection, as there is a possibility that there would be no need for the major operation. After blocking has failed to render permanent relief, there is time for surgery. Blocking the gasserian ganglion or the maxillary and mandibular divisions is a surgical procedure, requiring skill in technic and thorough knowledge of anatomy.

1008 West Sixth Street.

DISCUSSION

Leroy M. S. Miner, Boston, Mass.: My discussion will be limited to three of the subjects developed by Dr. Smith: (1) classification of trigeminal pain; (2) diagnosis, and (3) limitations of alcoholic injections in treatment of trigeminal neuralgia. In classifying the two types of trigeminal pain, it seems to me that the nomenclature is simplified and made more direct if we use the terms primary and secondary neuralgia. Primary neuralgia is the true tic referred to by Dr. Smith as the spasmodic or paroxysmal type. Secondary neuralgia includes those cases referred to as typical and arising from various etiologic factors that can be traced. Of utmost importance in trigeminal pain is a correct diagnosis; otherwise, treatment may be futile, for the treatment of a primary neuralgia differs widely from that of the secondary type. In an effort to make a diagnosis in these cases of trigeminal pain, a careful case history with such physical and laboratory examinations as may be necessary is important. If special attention is paid to the neurologic phases of a physical examination, illuminating information may be disclosed. The following case briefly cited illustrates this point: A man, aged 45, had for several weeks suffered with intermittent attacks of pain located in the second division of the right trigeminal nerve. Some of the teeth had been removed from the upper jaw and the maxillary sinus had been opened and explored, with negative findings. A short period of relief followed each operation, but each time the severity increased with renewal of pain. The physical examination at the time I saw him revealed three significant findings: (1) the knee jerk was absent; (2) the pupils were slightly unequal, and (3) areas of paraesthesia were found on the right side. These findings, coupled with a history of impotence starting about the time of the onset of the pain, suggested brain tumor. This diagnosis was confirmed. At operation, the tumor was found to be well advanced. The patient died ten days later. One cannot help considering whether the result would have been modified in any way had the case been diagnosed earlier. Occasionally, it is difficult to decide at once whether the case is a primary or secondary type. But before treatment other than palliative is instituted this point must be determined. I agree in general with Dr. Smith's statement regarding evulsion or neurectomy, but in my experience, a neurectomy occasionally gives long periods of relief. I agree that alcohol ordinarily should be used in treatment before resorting to resection of the sensory root, but we must recognize the fact that, even in most skilful hands, alcohol fails completely in a certain percentage of cases. It is but fair for the patient or the patient's family to understand this. Dr. Smith's development of technic is interesting and commendable. Every operator develops his own individual refinements of technic, but to Dr. Smith we are indebted for real advances made in management of the fifth nerve.

POSTURE AT THE DENTAL CHAIR AS RELATED TO THE DENTIST'S HEALTH

By ARTHUR H. NOBBS, M. S., D. D. S., San Francisco, Calif.

THE efficiency of the individual depends to a considerable extent on the way in which the body is used. If it is used correctly, and when it is in the normal state of health, he should be able to perform his daily tasks with the minimum amount of strain or friction; in others words, the body can return the greatest physical efficiency when it is properly used. When we depart from the normal in using the body, there is a waste in energy and a lessened efficiency on the part of the individual.

In order to develop an efficient body, it is essential to develop habits of good posture and to follow the fundamental principles of correct body use. It is more important to conserve the bodily force by adopting a favorable working position at the dental chair than to attempt to correct bodily defects by play or exercise. It is not intended to depreciate the value of either exercise or play, but rather to emphasize the value of exercise as a preventive rather than a corrective factor. Good body mechanics means the correct poise and control of the body with the normal functioning of every organ.

The practice of dentistry tends to strain the stabilizing and supporting framework of the body, which often results in a complete or partial exhaustion of the postural muscles from the pull of gravity. This generally results in a more or less serious derangement of the viscera, which frequently manifests itself in obscure and distressing ailments in later life.

The modern dental chair permits of such a range of adjustment that, by careful attention to its possibilities, a satisfactory working position can be secured which will do much in diminishing the dangers of faulty posture.

It is the purpose of this paper to call attention to some of the common errors of posture on the part of the dentist in order that much of the fatigue, and other attending sequelae, may be avoided.

In certain individuals, faulty posture may be caused by some congenital defect in the body mechanism, but in a large majority of cases, it is an acquired physical defect and it contributes in a large measure to both physical and mental strain.

Defective vision, flat feet, affections of the spinal column and other similar physical defects should be considered as important contributory factors, and should be corrected if proper posture is to be maintained while the operator is working at the dental chair.

When the body is not in proper mechanical position, it must function with some of its vital parts out of alinement. Correct posture favors health and promotes the highest physical efficiency by permitting the most effective functioning of the circulatory, respiratory and di-

gestive systems. To a certain extent, the human body can compensate for the strain caused by postural defects, but after a period of time, annoying and distressing symptoms often appear, which suggest the consideration of posture as an important contributory factor. Backache may be due specifically to poor posture, causing a strain on the supporting muscles and ligaments of the spine. Constipation may also be caused directly by poor body mechanics, in which case the relaxed abdominal walls do not support the stomach and intestines adequately and a partial stasis of the gastro-intestinal tract may result. Correcting the posture may raise the abdominal viscera to their correct position, thus improving the functioning of the entire digestive system. An incorrect adjustment of the dental chair may result in poor illumination of the field of operation. This compels the operator to assume an unfavorable working position, which results in eye strain, fatigue and numerous other conditions.

Good body posture will not cure all of the complaints enumerated above, but in the absence of a specific organic cause, faulty posture should be eliminated. In any event, an improvement in the posture will do much to increase the reserve energy so necessary in the successful conduct of a dental practice.

The question of fatigue is of major importance in considering the posture of dental operators. Fatigue may arise in the central nervous system, in the muscular system or in a combination of both. It may be manifest in a decrease of ability to continue work at the chair or in a decrease in the quality of the work accomplished. Much attention has been given to fatigue toxins, and it is reasonable to suppose that a posture that interferes with the proper circulation of the blood, especially the venous circulation in the abdomen, will contribute to fatigue, especially when this posture is repeatedly maintained for varying lengths of time.

Surveys relating to posture and fatigue have been conducted among several of the important industries. The reports of these surveys emphasize the beneficial results to be obtained by permitting brief changes in posture, which results in the promotion of the circulation of blood to the fatigued muscles. A similar procedure would doubtless be beneficial after a long continued operation at the dental chair. A brief change in position would promote circulation of blood to the fatigued muscles, and thereby contribute to the efficiency of the operator.

Correct posture distributes the weight of the body in such a way that there is an even strain on the skeletal and muscular framework. This becomes a most important factor to the dentist who, in later life, shows a tendency to put on weight. As so often happens, the additional weight appears in the region of the abdomen and very seriously interferes with the proper balance of the body. There is especial need under these circumstances to consider the posture of the supporting framework in order that serious complications may be prevented.

It is not the purpose of this paper to attempt to prescribe, except in a general way, correct posture for dental operators. References will be given which contain the most recent advice on that subject, and which may be easily obtained by those who are interested. After all, it is a relatively simple matter to adopt a favorable working position. The adjustment of the chair should be the starting point. The chair should always be adjusted so that the patient is comfortable, the operation can be carried

through in the most favorable manner and, more important, the operator will be subjected to the minimum amount of mental and physical expenditure. The feet should be firmly placed on the floor a slight distance apart. This will permit the maintaining of an erect position for comparatively long periods of time, with little exhaustion, and will make possible an easy balancing of the body. Many operators form the useless and unhygienic habit of resting one foot on the pedal of the chair while operating. This throws the body out of balance, necessitating an unnatural curvature of the spinal column and causing a general congestion of the circulatory organs. It invariably results in early fatigue and diminished working efficiency.

The proper placing of the foot control prior to its use will enable the dentist to maintain the most favorable working position, and still permit of the use of the dental engine. Too often, when the moment for use of the engine arrives, there is a blind reaching with one toe for the controller pedal and, during the period of its use, the body is entirely out of balance, the operator standing on one foot and in an unsteady working position. If the controller is placed in a favorable position before it is needed, due respect being paid to the optimal working position of the body, with the feet properly placed to maintain the balance of the body, it can be used without changing the position of the feet to any appreciable degree.

Most dentists are compelled to follow their chosen work for many years. In order to fulfil the highest professional responsibility to themselves and to their patients, they should make it a matter of great concern to maintain at all times the most favorable conditions of health. It is one of the obligations that they assume when they enter the profession and is important from the moral and economic as well as from the professional standpoint.

It is our hope that the young men just entering on their professional experience will form proper habits of posture at the dental chair, and that the older practitioners will find something of interest as well as of benefit in the points emphasized.

SELECTED REFERENCES

The following works are worth perusing by those who desire further suggestions on the subject of posture:

Fisk, E. L.: Health Building and Life Extension, New York: The Macmillan Company, 1923, pp. 237-259.

Thomas, L. C., and Goldthwait, J. E.: Bodily Mechanics and Health, New York: Houghton Mifflin Company, 1922, pp. 23-37.

U. S. Department of Labor, Children's Bureau, Bureau Publication, No. 164, Washington: Government Printing Office, 1926.

Bancroft, Jessie H.: The Posture of the School Child, New York: The Macmillan Company, 1918, pp. 102-108, 111-118.

CLEFT PALATE*

By WILLIAM L. SHEARER, A.B., M.D., D.D.S., F.A.C.S., F.A.C.D.,
Omaha, Nebraska

THE types of cleft palate that I wish to discuss are looked on as borderline cases which are seen rather late for successful correction. Class I embraces those cases showing delayed correction of a complete unilateral cleft of the hard and the soft palate associated with complete cleft of the lip and the nostril. Class II cases show a very broad cleft of the soft palate extending into the hard palate about midway between the posterior border of the horizontal plates of the palate bones and the lingual surface of the front teeth.

In the early years of my practice, I had many Class I cases. Most of the patients were from 5 to 17 years of age.

We know that any form of plastic surgery must have some kind of foundation, or at least a semblance of a foundation in order that the cases may be successfully handled. These patients are either too old for orthodontic treatment or, if they are not too old, the time required to bring about a result is too long for most parents to consider. Perhaps many of the cases could be quite successfully managed by orthodontia if they could be kept under observation long enough. In my experience, this suggestion has not been well received. Therefore, some sort of radical, surgical intervention, looking forward to a satisfactory result, has had to be considered.

Brophy gave us the greenstick fracture procedure, which, in many cases, is very successful. In this operation, the fracture is made in the long portion of the anterior ridge of the palate bone. Work is done somewhat in the dark, and one cannot see just how the fracture is progressing and where it will end. From the alveolectomy flap principle, I conceived the idea of laying back the mucoperiosteal flap, taken from about one-fourth inch above the cemento-enamel junction of the teeth, and carrying the flap up as high as was desired, sometimes to the base of the orbit. When the bone is so exposed, we can make the fracture exactly where we want it, respecting the pericementum of adjoining teeth. Also, the fracture can be guided laterally just where we wish, and thus fracture of the malar bone into the orbital cavity can be avoided.

When this is done, we can proceed to place silver wire through the two halves of the malar bones, using the Brophy straight-needle drill, as we would if the operation were performed in early infancy. With this open view method, we see exactly where we are placing the wires. The mucoperiosteal

*Read before the Section on Oral Surgery, Exodontia and Anesthesia at the Sixty-Ninth Annual Session of the American Dental Association, Detroit, Mich., Oct. 26, 1927.

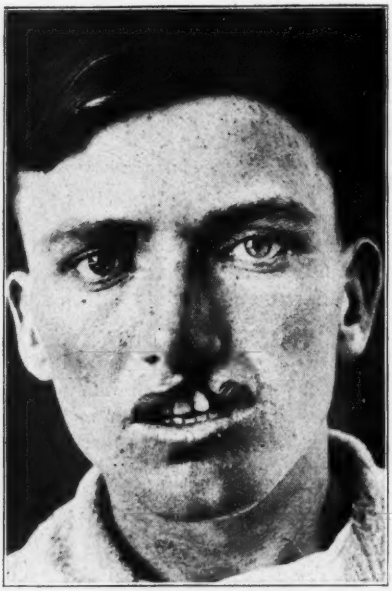


Fig. 1.—Patient before operation.

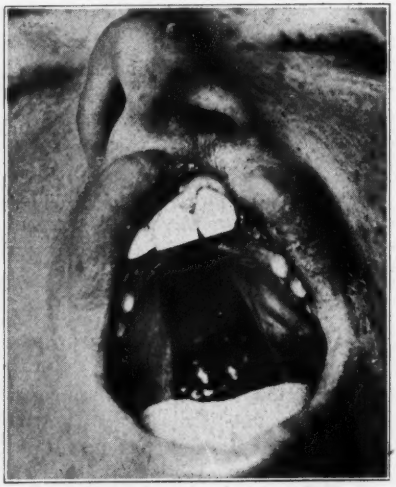


Fig. 2.—Cleft of palate, which measured 1¾ inches at the posterior border of the horizontal plate of the palate bone.

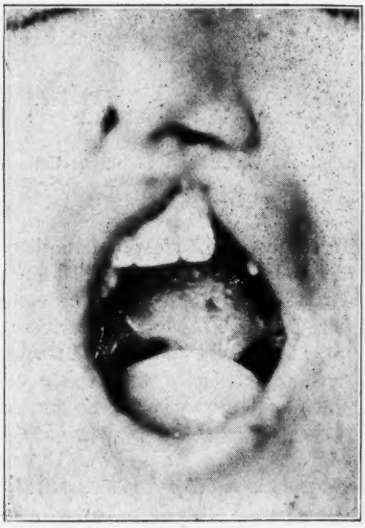


Fig. 3.—Closure of palate in one operation.

Fig. 4.—Palate after closure.

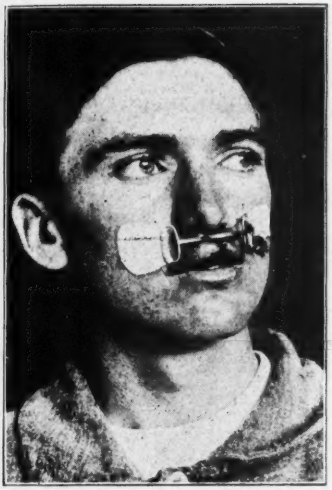


Fig. 5.—Logan traction bow, keeping tension from the suture line.

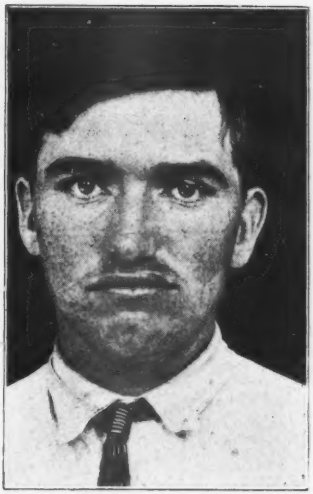


Fig. 6.—Appearance of patient one week after the sutures were removed.

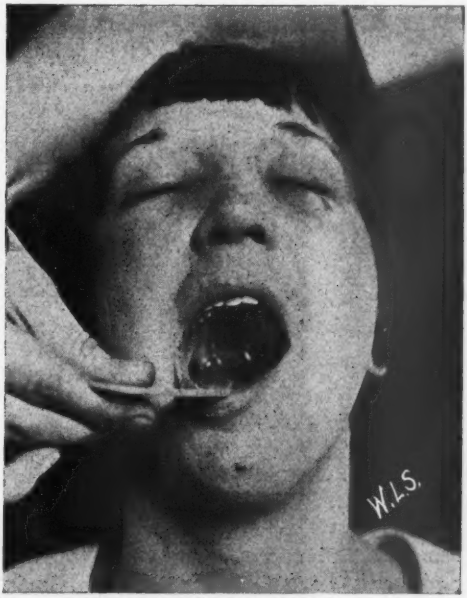


Fig. 7.—Cleft of palate before operation. The cleft measured 1¾ inches at the posterior border of the horizontal plate of the palate bone.

flaps are brought back and sutured into position with horsehair.

In laying back the flap, the distal incision of the flap is made somewhat distally to the line of fracture; the mucoperiosteal flap covers perfectly the line of fracture, and the secretions of the mouth are kept away from exposed bone surfaces. In former methods, sloughing of the tissue could not be avoided. In one year, I operated in eleven such cases, following the above-described technic. All healed by first intention.

This procedure has five major advantages: 1. We can see what we are doing. 2. The fractured point is completely covered with mucous membrane. 3. We have moved the nose into the middle of the face and corrected, at once, a terrible deformity. 4. We have obtained primary union. No sloughing or complications are liable to occur if the technic is carefully carried out. 5. We have closed the cleft of the anterior ridge and made a foundation for further plastic surgery, and also made it possible to restore, to a great measure, normality and speech function.

Class II cases, considered in this paper, represent those just on the borderline of possibility of correction. These cases, coming in as they do later in life, with atrophy of the muscles of the palate and throat, due to lack of full function, are very difficult, and often complications, by sloughing, occur.

In presenting these cases, I wish only to stress the importance of carrying out, in detail, the steps of the operation as

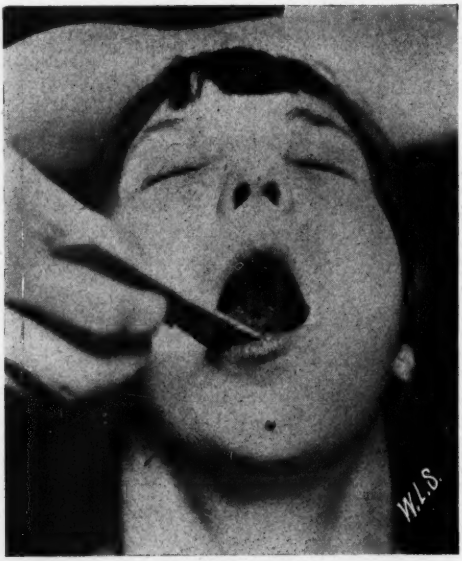


Fig. 8.—Appearance of palate after operation. Closure was made in one operation.

Fig. 9.—Cleft of bones in a boy, aged 5. A cleft of this character should be operated on at the age of from 3 to 6 weeks. Attempts at closure had been made before the author was consulted. In cases of this character, it is either necessary to employ a greenstick fracture technic or long slow movement of the jaws into position.

Fig. 10.—Condition after employment of greenstick fracture technic to close cleft (Fig. 9), and bringing of the nose into the median line of the face.

Brophy has given them to us, using silver wire, lead plates and horsehair, with the additon of an over and over grapevine stitch, which adds materially to the strength and, in many cases, I believe avoids sloughing coaptation stitches. It has been my experience that the more one packs with gauze of any kind, the greater the likelihood of complication and failure.

Fig. 11.—Appearance of patient (Figs. 9-10) after correction of lip.

ATTACKING "ACID FACTORIES" IN THE BLIND ALLEYS OF THE MOUTH*

By CARL PFANSTIEHL, Highland Park, Ill.

THE prevention of decay of teeth, rather than their repair, is now recognized as the most urgent problem in dentistry. The repairing of teeth lies wholly in the hands of the dentist, but to keep them healthy demands the intelligent and active daily cooperation of the individual himself.

Dr. Sullivan,[1] in a recent address, said, "We are told that 84 per cent of all children at the age of 3 have caries. A few years later, 95 per cent of the same children are virtually dental cripples." Dr. Bödecker[2] tells us that "according to even optimistic statistics, only two persons in a hundred are free from dental caries." No other branch of medicine pays greater dividends on an "ounce of prevention" than oral hygiene; yet the average citizen, intelligent in most matters of health, is comparatively ignorant as to the cause of caries and how best to prevent it.

The immediate cause of tooth decay is now recognized as what might be called "spot acidity."[3] Certain acid-producing bacteria, notably *Bacillus acidophilus*,[4] along with tiny fermenting food particles, are caught and sealed against tooth surfaces by an adhesive gluelike substance present in saliva and known as bacterial or gelatinous plaque, or simply as film. This substance, being insoluble in mouth fluids, prevents the trapped bacteria and food particles from mixing with the saliva, and so lactic acid is steadily produced, molecule by molecule, and held for days or weeks against one spot of enamel. Finally, when the strength or concentration of the acid at that point becomes great enough, the enamel is eaten away and decay is started. A few drops of saliva could neutralize all of the acid produced in one such isolated spot *if it could reach it;* but, unfortunately, the mouth fluids cannot penetrate

*This article is a continuation of the study recorded by the same author in a paper read before the Lake County Dental Society and published in the April, 1923, issue of THE JOURNAL.

1. Sullivan, E. F.: Children's Dentistry, J.A.D.A., 15: 176 (Jan.) 1928.

2. Bödecker, C. F.: Enamel Fissures, Their Development, Form and Diagnosis, Dent. Digest, 33: 78 (Feb.) 1927.

3. Robinson, J. B.: Some Considerations in the Study of Dental Caries and Its Treatment Looking Toward Tooth Conservation, Dent. Cosmos, 69: 181 (Feb.) 1927. Calloway, E. C.: Acid Mouth, Dent. Cosmos, 66: 242 (Feb.) 1924.

4. Bunting, R. W., et al.: Further Studies of the Relation of Bacillus Acidophilus to Dental Caries, Dent. Cosmos, 68: 931 (Oct.) 1926.

the film. Saliva itself can never become strongly acid enough to attack tooth enamel.[5] It is only this "spot acidity" that can and does.

The fact that over 90 per cent of all cavities are found between the teeth or in crevices shows that film with its little "acid factories" is not ordinarily removed from such hidden surfaces, even by the daily use of the toothbrush.

Gelatinous plaque also may be the basis of dental calculus or tartar. Under certain local or systemic conditions, mineral salts are precipitated in the film, which then gradually hardens into unsightly and gum irritating deposits.[6] Plaque is not confined to tooth surfaces only. The gums and soft tissues of the mouth are often more or less coated with an adhesive glutinous deposit which tends to trap bacteria and food particles and to favor localized acid fermentation in the folds and pockets of the mouth. This is particularly troublesome in the mouths of persons who have thick, stringy or ropy saliva because salivary circulation is retarded. Nature intended normal saliva to be relatively thin and watery so that the several pints that flow into the mouth daily could keep the folds and pockets washed free of food particles.

Modern civilization has changed our diet from hard, fibrous natural foods that require vigorous chewing and salivary lubrication, to our present soft overrefined and sugary foods. This change has taken place faster by hundreds of years than Nature could adapt our bodies to the new conditions. Chewing present day foods does not, as a rule, stimulate the circulation in the gums and tissues surrounding the teeth sufficiently to keep them healthy, or even demand enough exercise of the jaws to insure normal development of the entire masticating apparatus in the growing child. An orthodontist is by "artificial means" widening the jaws of both my small sons to make room for their permanent teeth.

Let us hope that some day changes will be made in human diet and habits of living so that Nature can restore our masticating apparatus to normal health and functioning; but, in the meantime, everything possible must be done to improve present methods of home mouth hygiene and to teach the general public the great importance of its diligent daily practice.

The principal object of home mouth care is to keep gelatinous plaques or film away from all tooth surfaces and soft tissues of the mouth. If this actually could be accomplished, it would prevent both "spot acidity" with its wholesale destruction of teeth and the formation of calculus or tartar, with its tendency to cause pyorrhea; and the natural beauty of the teeth would never be clouded. But, unfortunately, the methods now generally practiced fall far short of accomplishing this result.

Biochemical research has shown this mucinous film to be a complex mixture containing albuminous matter, mucin-

5. Reeves, H. G.: The Relation Between the Hydrogen-Ion Concentration of the Medium and the Decay of the Teeth, Brit. Dent. J., 49: 114 (Feb. 1) 1928.

6. Marshall, J. A.: Diseases of the Teeth, Their Diagnosis and Treatment, New York: Lea & Febiger, pp. 215-220.

ates and organic bacterial débris in an extremely tenacious and adhesive form. It somewhat resembles the thick nasal mucus discharged from the nose during a common cold. This viscid substance firmly adheres as a thin film to all tooth surfaces, particularly in the crevices between the teeth. It is insoluble in mouth fluids. Mild alkalis, such as soap solution, favor the formation of mucinates in the film, which makes it still more tenacious and adhesive, while the detergent action of soap, so effective on most objects, leaves the film undisturbed. Professor Gies puts it in this way:[7]

> The bristles of the toothbrush, when the latter is applied to the teeth in the ordinary act of brushing, are apt to pass through such a viscid film, after it is changed by alkali, without dislodging it; and the bristles may not completely remove the viscid mass, even by vigorous use of the brush. Anyone who has tried, with the aid of lime water or soap, and friction, to wash mucinous matter from a handkerchief containing nasal mucus knows how difficult it is to remove all the mucin from its attachments, under such conditions. The purpose to remove such mucinous material from the linen requires special effort when alkaline reagents are employed, apparently solely because of the adhesiveness of the viscid mucinate produced from the mucin by the applied alkali.

Strong alkalis might slowly dissolve the film, but they cannot be tolerated in the mouth. Mild food acids, such as are found in pineapples, oranges and apples, *if applied to the film,* tend to convert the mucinates into the less adhesive mucin and thereby favor the precipitation of the film. When this biochemical fact was first recognized,[7] several years ago, a number of tooth pastes were made mildly acid and offered to the public under the claim that their acid reaction would precipitate the film, but the reason for their apparent failure to accomplish this in practice is now recognized. It may be explained as follows: Saliva has a strong tendency to maintain itself in a practically neutral condition. When a mildly acid tooth paste containing less than 0.5 per cent of citric or tartaric acid, for instance, is taken into the mouth, the saliva begins immediately to neutralize the acid, and it quickly becomes too weak to be effective or is completely neutralized long before it can work its way through the saliva and reach the film, particularly in hidden crevices. Now, if a tooth paste should be made more strongly acid to begin with, in the hope that the acid would overpower the neutralizing effect of the saliva long enough to permit it to reach the hidden film in sufficient strength to be effective, it could not be tolerated in the mouth, and would probably attack tooth enamel. Under practical conditions prevailing in the mouth, it seems, acids must be either too weak to be effective on the hidden film or too strong to be initially safe.[8]

Abrasives in all degrees of harshness have always been used for cleaning teeth. Some dentrifices widely used today contain abrasives that are harsh enough to scratch the teeth, and if they are used daily for a period of years, the enamel may be worn through in spots. This destructive effect is seldom noticed by the user until it is too late. Tooth

7. Gies, W. J.: The Dentifrice Problem, J.A.D.A., 8: 851 (Oct.) 1921.

8. Pfanstiehl, Carl: The Chemistry of the Dentifrice Problem, J.A.D.A., 10: 301-316 (April) 1923.

enamel differs from all other body structures in that it cannot repair itself when even slightly injured. Abrasives, either mild or harsh, cannot be depended on to remove the film from the many little crevices hidden away between the teeth even through the vigorous manipulation of the toothbrush. Abrasives are effective on hidden surfaces and safe only in the skilled hands of the dentist.

Modern opinion agrees that to sterilize the mouth by the application of a medicated tooth paste twice a day is not only impossible but would be undesirable. Leonard and Feirer[9] recently examined forty-one different tooth pastes purchased at various drug stores and failed to find one capable of having any practical antiseptic effect on the bacterial flora of the mouth. In fact, they made the curious statement that all the pastes examined showed "a substantial *increase* in the average bacterial content of mouth rinsings during the first hour after scrubbing."

All this does not mean that it is useless to brush the teeth. On the contrary, it serves to emphasize the need for an even more thorough and careful brushing technic including the soft tissues of the mouth,[10] morning and night and as often as possible after meals. It is also a warning against depending on any dentrifice or toothbrush as a substitute for regular and frequent visits to the dentist for inspection and treatment if necessary.

A new and promising method of combating film on hidden as well as exposed tooth surfaces and on the gums and soft tissues of the mouth has recently been made possible through the peculiar physicochemical activity of a rare substance, galactonic lactone—heretofore known only as a chemical curiosity. It is an excellent illustration of the new interpretation now being given to that fascinating branch of science known as colloidal chemistry, the child of the union between chemistry and micromechanics.

Colloidal chemistry recognizes the purely mechanical activity which sometimes accompanies certain atomic or molecular rearrangements. It also deals with the mechanical activity of tiny particles of matter which are below microscopic visibility yet hundreds of times larger than ordinary molecules, as they bounce ceaselessly around in the fluid suspending them and in apparent defiance of the law of gravitation. Tremendous strides toward a better understanding of biologic activity are now being made in research laboratories through new applications of these principles.

Galactonic lactone is made by the hydrolysis and limited oxidation of the rare sugar galactose. The lactone itself is not a sugar and cannot be called a drug. It is perfectly harmless to the teeth and soft tissues of the mouth and is practically without taste. It owes its unique ability to attack and crumble the mucinous deposits under practical mouth conditions to the joint effect of two reactions, one chemical and the other

9. Leonard, Veeder, and Feirer, W. A.: Oral Hygiene (An Examination of the Alleged Antiseptic Properties of Toothpastes), Dental Cosmos, 69: 565 (June) 1927.

10. Stillman, P. R.: Stimulation of the Gingiva, J.A.D.A., 15: 1077 (June) 1928. Home Care of the Mouth, J.A.D.A., 15: 1367 (July) 1928.

micromechanical, which take place simultaneously.

The chemical part of the reaction may be explained briefly, as follows: Galactonic lactone is a neutral substance, but when it is taken into the mouth, some of its molecules tend to combine with the elements of water and become transformed into molecules of a mild food acid, namely, galactonic acid. The instant the surrounding medium becomes mildly acid, the conversion of the neutral lactone to galactonic acid automatically stops, and no more acid is or can be produced until that already present is used up or neutralized. Now, as fast as the mild galactonic acid so produced is used up, *but no faster,* more acid is released to take its place; and so this automatic replenishing of the mild food acid is carried on throughout the saliva, in every fold and pocket of the mouth and in the hidden crevices between the teeth, until finally all the galactonic lactone has been converted into the food acid. This is a process vastly different from taking a dose of acid directly into the mouth. It is more like the eating of an apple by a series of many small bites, with the exception that the food acid is progressively created in the mouth itself. This may be spoken of as a p_H controlled reversible reaction and is characteristic of a small group of rare chemicals known as the sugar-acid-lactones. The equilibrium point, that is, the strength of the acid (hydrogen-ion concentration) at which the conversion of the neutral lactone to its corresponding acid stops, is not the same with all the lactones, but galactonic lactone is particularly desirable because, if it were properly incorporated in a dentifrice for daily use, the maximum strength of the acid in the dentifrice could not at any time possibly exceed the acidity of an ordinary sweet apple. Galactonic lactone is, in effect, a potential and not an active food acid.

Now, as to the micromechanical phase of the reaction: A glance at the formula of the galactonic lactone shows the presence of a structure known as a "lactone ring" attached between the first and fourth carbon atoms of the chain. At the instant a molecule of galactonic lactone is converted into a molecule of galactonic acid, the lactone ring is disrupted and the general effect is to produce a specific colloid or, in other words, a localized mechanochemical disturbance, with results somewhat resembling a submicroscopic explosion.

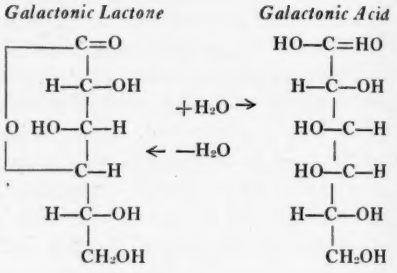
When galactonic lactone is applied properly to tooth and gum surfaces, the reactions as above described begin. Some of the lactone molecules react in the saliva, while some are swept against and are caught by the mucinous deposits, including those in hidden crevices, and react in the film itself. The effect of the mild galactonic acid so *released in the film* is to withdraw the basic elements from the adhesive mucinates and thus rob the mass of its adhesiveness, while the colloidal or mechanical activity acts as a specific detergent to break up or crumble the film, which is then more easily flushed away.

The mucinous material responsible for thick viscid saliva is precipitated in the same way, such saliva being rendered thin, and countless little food particles released from their attachments to the soft tissues of the mouth; and acid fermentation is thus attacked at

its source. The normal characteristics of saliva are not altered and its neutralizing power is not taxed beyond the healthful stimulation which accompanies the eating of a ripe apple or an orange.

Galactonic lactone was first suggested some years ago by Levene[11] as a possible substitute for strong acids in the cleansing of root canals. A few grams were made at great expense and used for this purpose with considerable success, but its complete physicochemical action was not understood or its application to oral hygiene recognized until several years later when it first became available in limited quantities for clinical experiments. The excessive cost of preparing galactonic lactone prevented its wider use, but very recently economical methods for its production have been perfected.

The pressing need for more effective methods and better practice for the daily cleaning of the mouth and teeth compels careful consideration of every attempt to find a solution of the problem, and the use of galactonic lactone appears to be a step forward.



11. Levene, P. A.: Part of the Report of the Committee on Practice—Dental Society of the State of New York, Dent. Cosmos, 63: 905 (Sept.) 1921.

THE SURGICAL ERADICATION OF PYORRHEA*

By A. W. WARD, D.D.S., San Francisco, Calif.

THE dental profession is forever seeking a panacea for periodontal lesions. Deep in the heart of the average practitioner, there lurks the belief that pyorrhea, like cancer, is incurable. But hope springs eternal that some miraculous drug or agent will one day be discovered which will banish the scourge. Dental history is replete with methods of treatment for the disease. For the most part, dependence has been placed on the alleged efficacy of germ destroying drugs. More than 100 different antiseptics and cauterizing medicaments have been used in treatment. Not one cure has been achieved through such methods.

Vaccines, stock and autogenous, have been tried by physicians, working alone or in conjunction with dentists. Scientists, with the aid of the microscopes, have endeavored to discover the bacteria

*Read before the Section on Orthodontia and Periodontia at the Sixty-Ninth Annual Session of the American Dental Association, Detroit, Mich., Oct. 26, 1927.

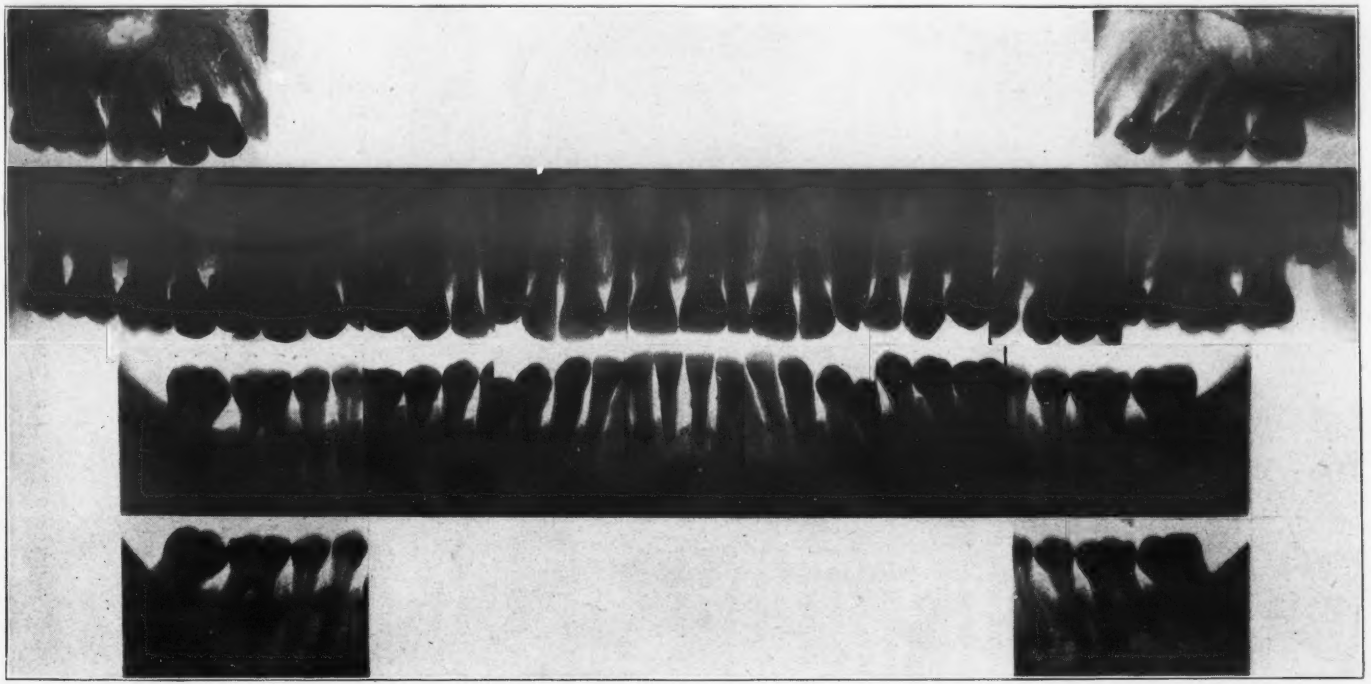


Fig. 1 (Case 1).—Roentgenograms taken in September, 1924. The patient had previously been advised to have all teeth extracted, not alone because of pyorrhea alveolaris, but also because of systemic disease attributed to the oral condition as a focus of infection.

responsible for it. Members of the medical profession, believing that dentists were unable to cope with the disease, have made some startling experiments and claims. Goadby reported cures by the use of autogenous vaccines. Experiments by others did not bear out his claims. Bass claimed to have found a specific in emetin hypochlorid. Subsequent experiments by hundreds of dentists have not brought to light the cure of a single case with emetin. Another physician invented a vacuum machine designed to evacuate the pus pockets and stimulate the soft tissues. This likewise

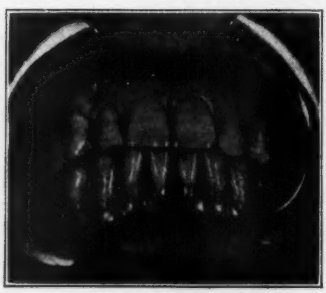


Fig. 2 (Case 1).—Appearance of the patient's mouth, September, 1928. The patient is in splendid health. No medical treatment has been necessary since the operation for pyorrhea, September, 1924. The teeth are tight in their sockets and the gums are healthy. There has been no scaling or treatments since the operation, but simply brushing and taping by the patient.

proved a failure, as did the injection of succinimid of mercury into the gluteal muscles. Mercury, iron, arsenic have all been used unsuccessfully, and now the new pyorrhea fad is diet. I have mentioned these failures for no other reason than to call attention to the fact that the problem of the eradication of this and other mouth infections has occupied the minds of physicians as well as dentists.

There have been reported too many recoveries from chronic disease in which no treatment other than removal of some focus of infection has been instituted, for the most skeptical to say that the cures were chance results. Dermatologists, psychiatrists, neurologists, rhinologists, pediatricians, ophthalmologists—all need the aid of the dental diagnostician and surgeon. Medical diagnosticians, specialists in the various branches of medical practice, and well informed general practitioners of medicine are thoroughly acquainted with the importance of infective foci in the mouth in relation to systemic disease. Billings, Rosenow, Duke, Moody and many others realize fully the significance of focal infection, especially in the teeth, as causative factors in the production of systemic disease.

In twenty-five years, new and better methods have been advanced in nearly every branch of dentistry. Desirable innovations have been introduced by the colleges and general practitioners as well as by the specialists in the various branches of the profession. Prosthodontists, orthodontists, exodontists have made great strides. Compare the oral surgery of the present with that of 1900. Compare the related esthetic dentures with the makeshift plates of the beginning of the twentieth century. Consider the adoption of Black's cavity preparation; the roentgen ray; the casting process of Taggart; procain anaesthesia; the Novitsky flap operation in oral surgery; the porcelain jacket crown and inlay; inlay abutments for fixed and removable bridgework; Tinker roots; steel facings; movable-removable bridgework. Consider, finally, the great good which has accrued through dentistry by the removal of infected and pulpless teeth.

But how much has the science of periodontology advanced? Are the uni-

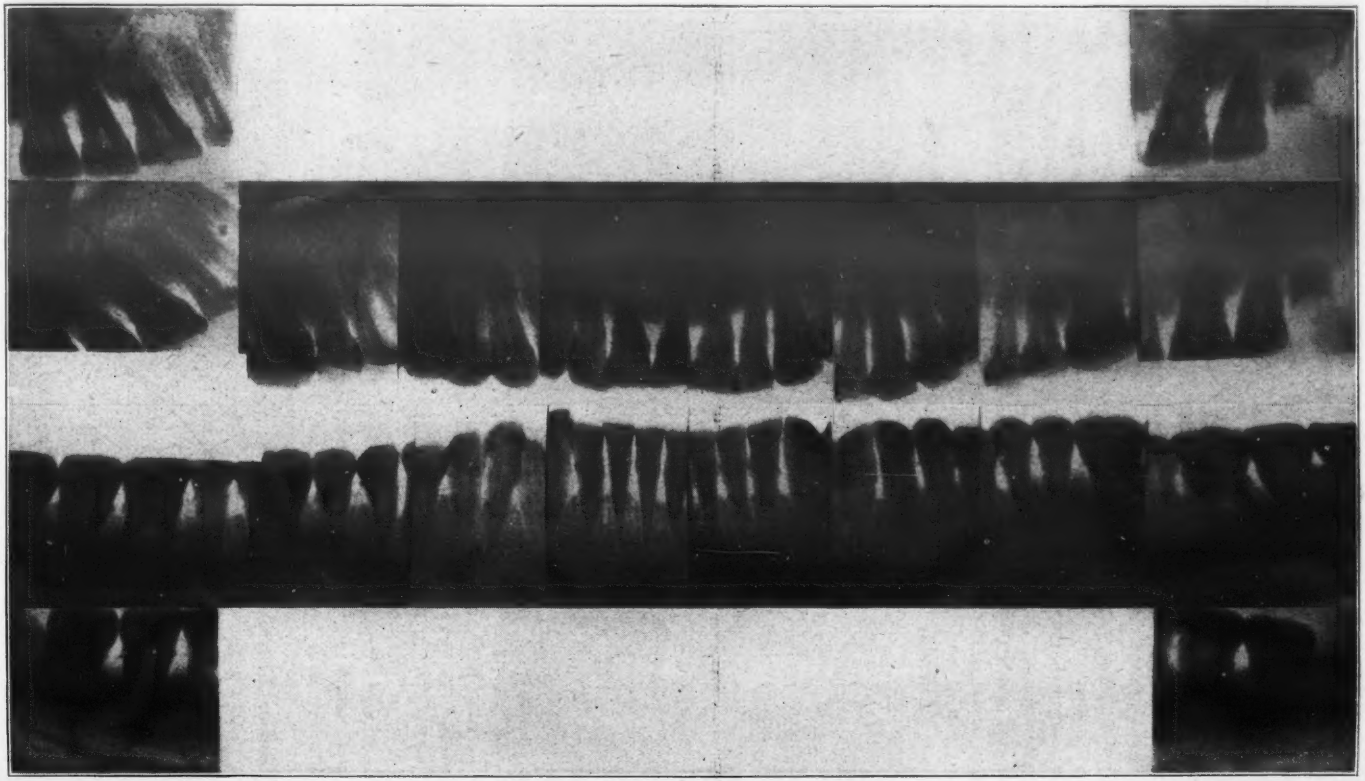


Fig. 3 (Case 2).—Roentgenograms taken in April, 1926. The patient had been advised by two dentists to have all teeth extracted. He had been ill for approximately one year prior to the operation.

versities teaching a rational method of eradicating disease about and around the roots of the teeth? Are the leaders of periodontology themselves really eradicating pyorrhea by scientific surgery? Or are too many of the schools, too many of the periodontists of note, too many of the foremost general practitioners continuing in the errors of their preceptors, scraping root surfaces covered by infected gum tissue, endeavoring by removal of the products of disease to eradicate the disease? This is exactly what is being done by 49,000 dentists at least, out of 50,000, and the results are not much better than they were twenty-five years ago.

Fig. 4 (Case 2).—Appearance of patient's mouth, September, 1928. The teeth are tight in their sockets and the gums are in a healthy condition. The patient's general health is excellent. There are no further medical treatment and no dental treatment except brushing and taping by the patient.

Dentists everywhere are looking to the periodontists for guidance. Physicians are sending patients to them for the removal of mouth infections. Doctors of dental surgery! A strange title—when all the surgery they do is the scraping of a few deposits of serumal calculus off of surfaces which they cannot see. (They hope that, by some miracle, the soft tissue will become attached to the root surface.) This would be amusing if pyorrhea were not a grave focal infection. On its successful eradication, the physician is many times depending for the cure or amelioration of chronic disease.

Known to be the most common source of systemic disease, pyorrhea can be the focus for at least twenty diseases. Arthritis and neuritis are so commonly cured by its eradication that, in many cases, it is a specific for these conditions, demonstrated hundreds of times. Heart lesions; cholecystitis; pancreatitis; nephritis; disturbances in the blood stream; ulcers, gastric and duodenal; labyrinthitis; infections in the sinuses or the tissues of the eye; joint disease—all respond to removal of primary foci in the mouth, provided these teeth and gums are the only foci and the damage to the secondarily infected organ or organs is not too great.

The tonsils and the maxillary sinus seldom remain healthy in the presence of long standing tooth and tooth investment disease. Because of continuity and the presence of the lymphatics, these organs become involved, and they sometimes remain sources of systemic disease long after the removal of tooth infection. It is well to remember that the teeth, the sinuses, the ear, the eye, the brain are intimately associated. It is impossible to foresee the results, or to measure the injury that may result, to a patient from tooth infection. Periodontoclasia is the most frequent infection. The diseased teeth infect the sinuses. The secretions pass over and infect the tonsils. The purulent material is swallowed or absorbed, causing gastric or duodenal ulcer, or spasm which simulates these conditions. Stimulation to the sympathetic nerves may cause pain in remote parts, the heart, etc.

All sinus disease is not brought about by infected teeth. But my clinical observation is that chronic sinusitis accom-

panies mouth infection too often to be a mere fortuitous occurrence. But this is only one of the routes, the least traveled. By metastasis, bacteria, toxins and filtrable virus travel to a point of least resistance anywhere in the economy. A secondary infection is set up which may or may not respond to the removal of the primary infection. This depends on whether or not there is more than one primary focus; the extent of the injury to the secondary; the virulence of the organism or virus; the interval of time elapsed between the secondary infection and the removal of the focus; the age of the patient, and the comparative resistance of the patient.

When a physician sends a patient whom he is treating for sinusitis, arthritis, neuritis, iritis or some other disease to a periodontist of the old school with a request that he immediately remove the source of the oral infection, the periodontist will say, "Yes, Doctor, send the patient to my office an hour a day for thirty days and I will scrape his tooth roots."

The physician has had just such ridiculous replies before. So he orders the patient to a hospital with instructions for an exodontist to extract the teeth. The physician is right. He would rather have a toothless patient than a crippled or dead one. But thousands of teeth are unnecessarily lost under these circumstances. It takes no longer to eradicate pyorrhea surgically than it does to extract the teeth. If pyorrhea cannot be cured in one sitting, it cannot be cured at all.

There are some obvious reasons why the surgical eradication of pyorrhea pockets brings about the most favorable results. In the process of healing, following surgical eradication, the alveolar process in the proximal surfaces is covered over by connective tissue. And this, if properly stimulated by brushing and massage, forms a tight ring about the neck of each tooth operated on, preventing reinfection. The removal of infected gum and periodontal membrane together with carious bone cells tends to

Fig. 5.—Progressive steps in the surgical eradication of pyorrhea alveolaris as originated and advocated by the author.

prevent further degenerative change. The destruction of the alveolar process in pyorrhea is many times a true bone caries. And unless the carious bone cells on the affected surfaces are removed surgically, the destruction of bone will continue, no matter what other procedures are instituted. Caries in teeth admittedly must be removed. Caries in bone in other parts of the body is removed surgically. What reason can be given for not removing it on the alveolar process, except ignorance and timidity? Gum tissue and tonsil tissue are histologically of the same type. In his efforts to abort or cure systemic disease, does a competent physician hesitate to remove diseased tonsils? If it is good surgical judgment to remove bacteria permeated tissue in the one case, why not in the other?

These conditions occur in pyorrhea: impeded circulation; caries of the alveolar process; absorption, or, sometimes, separation of the pericementum from the cementum; hypertrophy or atrophy of periodontal membrane beyond the actual pocket due to disturbance of circulation; infection and trauma; changes in the dental pulp due to the same conditions; inharmony or malocclusion brought about by movement of a tooth or teeth, the movement caused by the thickening of the abused membrane. Hence, regardless of all other factors—esthetics, fear of the patient, timidity of the operator—pyorrhea pockets must be eradicated. By eradication, I mean cutting down or removal, so that the teeth, the mouth, the individual will be restored to health even though the thought of such radical procedure shocks the delicate sensibilities of the finer souls in the field of periodontology. Infection in the mouth must be eradicated.

The same rule should apply as in cancer. Operate early. Many dentists who believe in surgery consider it necessary for pyorrhea to have reached the almost incurable stage before they resort to surgery. It is good practice to remove caries from tooth structure in its incipiency. Likewise, it is true that the sooner the carious bone cells in a periodontal lesion are removed, the better. Remove pulpless teeth, eradicate areas open to secretions of the mouth whether they are suppurative or not, unless acutely infected, in which case, as in Vincent's infection, attack first the specific organisms.

This rule applies as well in periodontoclasia as it does in the removal of gallbladders; in prostatectomy; in tonsilectomy; in radical surgery anywhere in the body. The scraping of root surfaces in an endeavor to eradicate foci of infection about teeth fails because, at best, only some of the results of the disease are removed. They are serumal calculus and infected cementum. Wherever there has been considerable alveolar absorption about the molars, the efforts of the best operators to remove the deposits and to freshen the cementum are unavailing. Even if all deposits were removed, if all denuded cementum were freshened, the focus of infection would remain. Unless the infected gum and infected alveolar border and detached periodontal membrane are entirely removed, the disease will progress and systemic infection persist.

Whether the condition or disease with which we are concerned is called periodontoclasia, pyorrhea, suppurative gingivitis or any other one of a hundred quasiscientific names, thousands of people in every clime have looseness of the teeth and disease of the gums sometimes accompanied by a flow of pus from the

tooth sockets. Whether or not the disease is brought about by heredity, systemic disease, faulty diet or inharmonious occlusion, the condition causes systemic disease. For this reason, it becomes the duty of our profession to combat it in the most efficient manner. Any and all procedures for the eradication of pyorrhea which have been found unsuccessful or partially so should be discarded for the most uniformly successful method.

For ten years, I conducted postgraduate classes in the Carr system. During this time, it was my good fortune to become acquainted with many of the foremost periodontists of America and to ascertain the degree of success which is obtained by these operators in methods known as planing and scaling of root surfaces. These methods have been the main reliance of skilful specialists throughout the country. Such operators have been a splendid adjunct to the profession. Their desire or aim has been the complete removal of all calcareous deposits on the root surfaces in an effort to freshen the cementum to the depth of the former attachment of the pericementum. This method and an endeavor to correct inharmonious occlusion has brought about some splendid results.

But it is too often unavailing for the following reasons: 1. The operator is unable to see the surface on which he is working. Dependence is therefore placed on the sense of touch because the calculus and infected cementum are covered with gum. 2. The gum tissue which is part of the infected pocket is permeated with bacteria. (Box.) Nothing is done to remove the infection within the gum. This invites recurrence. 3. No care or operative measure is given to the alveolar process surrounding the affected tooth roots or the detached periodontal membrane. Roentgen-ray examinations will reveal the fact that surgical interference is called for, especially in the proximal spaces. This is denoted by a dulling of the apex of the alveolar triangle in the proximal space. To the depth of the rarefaction, the bone is actually carious, and the condition should be treated as caries in a tooth or caries in bone in other parts of the body.

There are three surgical operations designed for the eradication of pyorrhea: (1) gingivectomy, the removal only of the unattached gum; (2) the flap method, in which the gums are detached labiolingually and flapped back in such a manner as to allow a view of the diseased alveolar process and root surfaces; and (3) the obliteration method. By obliteration, I mean a procedure which does away with those structures which constitute the pocket; that is, all soft tissue above on the lower teeth, or below the alveolar crest on the upper, when diseased and detached, is to be removed in its entirety. Most important of all, there must be removed a layer of diseased bone which is impregnated with bacteria. This surface should be left smooth so that the regenerating fibrous tissue will have a compatible foundation.

The flap method falls far short. If the part of the gum tissue and detached periodontal membrane constituting the pocket is not removed, the area is covered again with infected soft tissue. And if the marginal or infected gum tissue is removed, the soft tissue must be stretched over the operative areas. Consequently, the freshened bone areas are covered by a tissue not compatible since it is not possible to stretch or pull over the tissues on the lingual surface of the maxilla because of their thickness and rigidity. If, on the lingual surfaces of the bicuspids and molars on the mandible, the infected soft tissues are cut away

after the flap is made, it would be necessary to denude the bone as far down as the floor of the mouth to obtain a slack enough flap to bring about apposition with the buccal flap. Hence, briefly, the flap operation can never be a success because of the inadvisability of making flaps on the lingual surfaces; the danger of hemorrhage; the hazard of infection; the unnecessary denuding of bone. Gingivectomy removes only infected gum and part of the infected periodontal membrane. Incurable cases are those in which (1) it is impossible to establish harmonious cusp and incisal relationship; (2) so much alveolar process has been lost that the nearly normal masticating of the individual cannot be tolerated; (3) multirooted teeth have lost the alveolar process in the bifurcations; (4) the teeth, owing to the structure of the alveolar process and the peculiarity of the bite, have undergone absorption of the apices or a pronounced thickening of the periodontal membrane at the apex, even though vital; (5) there are pulpless teeth; (6) the teeth are so placed in the arch that their roots are in apposition for a considerable distance; (7) teeth with large bulbous crowns and abnormally small short roots have lost a considerable amount of alveolar support and so are very bad risks; (8) any tooth has lost too great an amount of bone support. Experience alone can determine the amount.

The operation which I have devised for the surgical eradication of periodontal lesions is a simplified technic which I believe fulfils all requirements. All cases are conducted exactly alike; that is, definite steps are taken in sequence. An especially designed instrument is provided for each step. It is unnecessary to view the operation in its entirety. The results are uniform. The operator can be sure that all diseased tissue has been removed at each step. Great speed is possible and certainly desirable. The convenience and comfort of the patient require the completion of the task in the least possible time. Two hours is sufficient for a skilful operator to eradicate all periodontal infections in any case. The last reason, but not the least, for the popularity of the operation is the absence of pain. When the wounds incident to these operations are properly cemented over, the great drawback to surgical eradication is surmounted. The operator can cover as much territory as his skill and time will allow without a thought of after-pain, shock, infection or hemorrhage, the deterring factors in any operation.

SUMMARY

Pyorrhea, the most common focus of infection, is the cause of systemic, chronic diseases, arthritis, neuritis and any other diseases caused by the invasion of streptococci and staphylococci.

The gums and alveolar process are the most common portals of entry because of the fact that in pyorrhea the wounds are open. There are sometimes as many as sixty pyorrhea pockets in one mouth, each harboring as much infection as the crypt in a tonsil. The tonsil is lymphoid tissue and combats invasion of bacteria. The alveolar process and gums are poorly supplied with lymphatic barriers to invasion.

Old methods are of no avail in the eradication of the disease because the infected gum and detached periodontal membrane left intact are a source of reinfection. My method is an improvement over former surgical methods because of the design of original instruments. It is possible to eradicate all pockets and thoroughly eradicate all pyorrhea in one sitting.

Formerly, surgical eradication of the disease was accompanied by postoperative pain due, first, to trauma and, secondly, to infection of the unprotected wound. My method is to cement over the wound with a specially designed cement or surgical packing placed on immediately after the operation, for the purpose of immobilizing the tissue just operated on, and to shut out the ever-present infective organisms. This packing overcomes all the objections to surgical eradication, pain, hemorrhage and infection. It is left in place ten days, and when it is removed, the alveolar process is covered with a new resistant soft tissue.

Pyorrhea is curable by surgical eradication, regardless of diet, if followed by strict oral hygiene and the adjustment of the bite or occlusion, and without the aid of drugs of any sort.

In conclusion, I wish to pay tribute to Arthur D. Black, who has drawn the attention of the profession to the necessity of surgically eradicating periodontal lesions and to the necessity, especially, of curettage of the alveolar bone; also to W. J. Charters for the development of his technic in brushing, which is a valuable adjunct to this work. I wish also to thank Shirley Ashby of San Francisco for the assistance he gave me in originating a better method of packing mouth-wounds.

DISCUSSION

R. J. McGavock, Birmingham, Ala.: In the lower mandible, where the gums are apparently tight around the anterior teeth, and you find bifurcation as you go between the teeth and there is a pocket that runs under the bifurcation and all the way through, what procedure would you follow?

Isador Hirschfeld, New York City: I understood Dr. Ward to say that "the men who adhere to the conservative method in the treatment of suppurative periodontoclasia, simply removing the products of infection, naturally must fail." As a rule, those of us who treat suppurative periodontoclasia by the conservative method, with but few exceptions, think in terms of more than the mere removal of the products of infection. We think primarily of the etiology and try to remove or remedy as many of the etiologic factors as we can recognize, whether they be the original or secondary factors. I also understood Dr. Ward to say that "these men merely scrape the teeth in the treatment of pyorrhea." This, I claim, is not so. He states that the process of disease in the bone is one of caries and therefore all the bone that has been at all infected or affected must be thoroughly removed. I wonder how he can know where the infection has stopped. Is it possible to remove the furthermost part of the bone and see that there are no bacterial organisms there? After all, is it not true that we cannot remove all infection, but that if we can raise the resistance of the tissues to normal and keep it so, we have done about as much as can be expected? Dr. Ward says that pyorrhea is the cause of systemic disease. While that may be true, is it not a fact also that pyorrhea may be the *result* of systemic disease, and if that is so, why not consider the systemic angles also instead of treating the periphery only?

H. T. Stewart, New York City: I see no objection to using the word "pyorrhea," and I should think that that monstrosity, "periodontoclasia," ought to be omitted from our nomenclature.

S. J. Rabkin, Cincinnati, Ohio: How would you explain the physiologic process of repair following the operation? Does a re-creation of cells take place in the hard and soft tissue? Is there a proliferation of cells, bone as well as tissue? Do you also believe that you have a source of bacteria absorbing and digesting these bacteria?

Dr. Ward (closing): My attitude in regard to periodontoclasia probably differs from that of some others in this field of endeavor. I am primarily interested in the health of my patients, working as I do with physicians and surgeons who are anxious to eradicate all foci of infection in the shortest possible time. Careful physical diagnosis will prove that all persons with pyorrhea of long standing have some physical defects caused by infective foci. Duodenal ulcers, gastric ulcers, heart lesions, diseases of the eye, overweight, underweight, disturbances

of the ductless glands, arthritis, nephritis, neuritis, pancreatitis, so-called nervous diseases, may all be caused by bacterial invasion from infective foci; consequently, the physician wants the foci eradicated now. He is not interested in any theory of reattachment of soft tissue to cementum. He knows histology well enough to know that it will not happen. I challenge any champion of the reattachment theory to show one case in which any soft tissue has reattached where a pyorrhea pocket has once existed. My challenge is issued after investigation in thousands of cases has not revealed a single so-called reattachment. In answer to the question as to what I consider the proper method of treating teeth where bone is lost in the bifurcations: I consider it safest, for the patient, to extract teeth so badly infected, especially the upper molars. Sometimes, lower molars with long wide spreading roots can be successfully operated on. Always, whenever successfully operated on, the mesial and distal alveolar process must be shortened until it is as low or lower than the bone in the bifurcations. In reply to Dr. Hirschfeld, I am assured by no less an authority than Dr. Moody that I am correct in my statement that bone destruction in a pyorrhea pocket is true bone caries. Dr. Hirschfeld asks how I know when we remove the caries that we have removed all infection. I know just as well as any surgeon knows when he operates on any septic wound. Wherever possible, surgeons are doing a débridemènt that is cutting widely into healthy tissues. This is one of the most important lessons taught by the late war. Thousands of lives were saved by this procedure in shrapnel, bullet and other wounds. Gas gangrene septicemia was prevented in thousands of cases. This has become common practice now, and such a dreadful thing as a carbuncle is innocent enough when the surgeon cuts far enough into healthy tissue. The United States Army Medical Department has named the pyorrhea eradication operation gingiva alveolus débridemènt. In this operation, we remove enough to feel reasonably sure that we are in uninfected tissue, and Nature is usually very kind. Sometimes, the cancellous bone in the approximal spaces is lower than the cortical plates. In these conditions, I remove enough of the plates so that the lowest point is level with or a little higher than the external bone.

HEALING OF TOOTH SOCKETS FOLLOWING TOOTH EXTRACTION IN DOGS*

By WARREN S. SCHRAM, D.D.S., St. Paul, Minn.

THIS is a preliminary report of a series of studies made at Northwestern University during the past year with the hope of obtaining some definite information regarding the process of repair following surgical interference with the maxillary bones.

The literature on general bone regeneration, which dates back to the early part of the eighteenth century, is voluminous. Practically all of the work was done on the long bones. The object of the various experiments made was to determine which of the cellular elements of bone, such as bone marrow, the cancellous bone or periosteum, was most active in bone formation, and whether any of them could be definitely excluded from the bone forming process.

Arthur Keith[1] sums up the varying opinions of these men as follows: "And

*Read before the Section on Histology, Physiology, Pathology, Bacteriology and Chemistry (Research) at the Seventieth Annual Session of the American Dental Association, Minneapolis, Minn., Aug. 22, 1928.

1. Keith, Arthur: Brit. J. Surg., 1919.

here we are in the twentieth century still with the same two groups, those who regard periosteum as the chief osteogenetic element of the human skeleton, and those who regard bone itself as that element." Where is the truth? As in most cases of this kind, there is truth on both sides. But the greater share is on Macewn's, whose conclusions are that periosteum has no osteogenetic function in itself but acts as a confining and delimiting membrane for the cells which are active in bone formation.

Our work deals primarily with the regeneration taking place in the alveolar process. In reviewing the English literature, we find that men writing on the subject of regeneration taking place in the maxillary bones have based their opinions on the findings reported from long bone experiments.

H. W. MacMillan[2] says, "Dental literature abounds with terms which describe the alveolar process as transitory and unlike true bone." And he states that, in his opinion, this tissue is true bone, differing only from the other bones of the body because of the demand of function. I refer to this controversy because of the direct affect which it might have on the problem of regeneration in these tissues following injury.

The work which I have done consists of the microscopic study of the process of regeneration taking place in the maxillary bones of dogs at varying intervals after tooth removal both by simple extraction and by surgical methods.

Dr. Euler[3] made a similar study of simple extraction wounds in 1923. Part of my work closely parallels and agrees in general with his.

Several dogs with sound teeth were selected. The upper and lower bicuspids were removed with simple extraction on one side and with surgical extraction on the other. The differences, if any, which would be found in the healing process were to be noted, the work being done with the same care in regard to technic and asepsis as would be used in human cases. At varying intervals, the dogs were killed, and sections from the operative areas were studied microscopically.

We were unfortunate in obtaining material from the wounds produced by surgical removal. Through accident, we lost the material for the 8-day, 2-week and 3-week specimens. It is evident from our slides of simple extraction that these would have been most interesting. One slide of a surgical removal of 3 weeks became infected, owing to the loss of the suture before initial healing had taken place, thereby exposing the alveolar bone to the fluids of the mouth.

(In this slide, there is evidence of the epithelium dipping down between the alveolar bone and the gum flap, thus seriously interfering with the healing process. The entire area of exposed bone is necrotic, and it is of interest to note that the destructive process has progressed to the pericemental membrane and dentin of the adjoining tooth, destroying small areas in the cementum.)

This, of course, is a condition which might occur in surgical removal or wherever the alveolar bone is left unprotected by a blood clot and exposed to the fluids of the mouth.

From the work which we have done, I believe that we have been able to study fairly accurately the regenerative process taking place after simple extraction,

2. MacMillan, H. W.: J. Dent. Res., 6: 251, 1924-1926.

3. Euler: Deutsch. Monatschr. f. Zahnheilk., December, 1923.

and our work corresponds almost entirely to that done by Dr. Euler. We have seen disease which may occur after the exposure of the alveolar bone to the oral fluids. As yet we are not prepared to make definite statements regarding repair following surgical removal of teeth. We hope to complete this work.

TREATMENT TO ENLARGE ABNORMALLY SMALL MOUTH FOR FULL DENTURE PROSTHESIS: REPORT OF CASE

By B. S. BURKS, D.D.S., Crewe, Va.

ABOUT three years ago, a patient came to my office for an extraction. On examination, I found the mouth to be greatly drawn, as were the hands and fingers. The extraction was done with a great deal of difficulty, owing to the smallness of the mouth.

The patient stated that, nine years previously, she was affected with a disease which caused drawing of the muscles of the mouth, hands and fingers. Her physician was puzzled, but said that the condition was due to some nerve trouble. Treatment gave no great amount of relief. On questioning the patient further, I found that the case was congenital to a certain extent. She left my office without my obtaining any further history.

In about a year, she returned suffering with odontalgia. On examination, I found a pyrroheic condition and so advised extraction of the few remaining teeth. After a time, the patient came in to have a denture made.

Now the real problem faced me. How was I to get into patient's mouth an impression tray which would be large enough to fit the inside of the mouth? A No. 4 impression tray could not be gotten in, and even if we had been able to insert it, it would have been too small for the arch. Day after day, I studied the case and finally sent the patient to Richmond to G. W. Holliday for an examination.

A few days later, Dr. Holliday wrote me that it was the most extreme case that he had ever seen and he did not see how it would be possible to make a denture for the patient in her present condition. He suggested some mechanical device to enlarge the mouth by stretching the muscles. I had the patient try a large marble, a bottle and several other devices, but with no success.

I then determined to attempt enlargement of the mouth by having the patient use her fingers to stretch the muscles. She was therefore instructed to put her fingers in the corners of her mouth and pull against the muscles for one hour and twenty minutes each day.

May 5, 1927, this procedure was begun, and the patient was instructed to report to my office every two weeks for examination. Soon I noticed the muscles had begun to relax just a little, and I felt greatly encouraged. In three months, there was a marked change, the expansion being very noticeable. The patient was as greatly delighted as I was when we realized that our goal was in sight. Three months later, the mouth had been expanded to such an extent that impressions of compound and plaster were successfully taken. The following is an extract from patient's letter.

"I have had my teeth for five months and can eat anything. I will always be more than grateful to Dr. Burks for his work and will be glad for any visiting dentist who comes to Crewe to examine my mouth."

I fully realize that had I been able to obtain a more complete history of the case, it would have been more interesting. But to have performed this service for a cooperative and appreciative patient was a source of great satisfaction.

The Journal of the American Dental Association

58 East Washington St., Chicago, Illinois

C. N. JOHNSON, Editor

Published by the American Dental Association

All expressions of opinions and all statements of supposed facts are published on the authority of the writer over whose signature they appear, and are not to be regarded as expressing the views of the American Dental Association, unless such statements or opinions have been adopted by the Association.

Communications intended for publication in the scientific or literary pages of THE JOURNAL should be addressed to the Editor. All advertising or business matters should be addressed to the Business Manager.

Subscription is included in the annual membership dues. To nonmembers living in the United States or its possessions, the price is $2.50 a year in advance; Canada, $3.00; Australia, $4.00; other countries, $3.25. All foreign subscriptions are to be paid in United States money.

Editorials

ENDOWMENTS TO DENTISTRY

The medical profession has been favored by philanthropists who have made liberal endowments for medical education and research. During recent years, these endowments have run into large figures, from 1910 to the end of 1926 the total aggregating more than $262,000,000, which is really a very encouraging contribution to science.

All of this is most commendable, and the hope is hereby expressed that, in the future, these endowments may be even more liberal than in the past. Anything that will further the physical well-being of the people is worthy of the highest commendation, and philanthropists can do no better service with their money than to devote it to this most worthy purpose.

But what reason shall be given for the fact that, in this wide distribution of funds, the profession of dentistry is so completely overlooked? The only possible reason is that the real significance

of dental service has never been adequately brought to the attention of our moneyed people. If our wealthy men only knew just what dentistry has done for humanity, and what it is capable of doing in the future if favored with the proper facilities, there is no question that endowments would come to it in abundant measure.

That the world owes to dentistry the discovery of the great blessing of anesthesia should in itself command the respect and gratitude of every man, woman and child in the universe. Try to conjecture what civilization would be without anesthesia, and see if dentistry is not entitled to some consideration when funds are to be donated for study and research. Dental service has been instrumental in curing many diseases that had baffled the physicians. It has added to the physical welfare and the beauty of countless numbers of people, and has robbed humanity of one of its most hideous deformities. A distortion of the human countenance is a distinct handicap to the prospects of any girl or boy, and a profession that changes a deformed face into one of harmony and beauty is entitled to the gratitude of the entire human family.

One reason why endowments have not come to dentistry more plentifully has been that it is a comparatively new profession, and has not made the spectacular appeal to philanthropists that medicine has. If it were only known that dentistry is capable of contributing concretely to the longevity of the race, and of making humanity more comfortable and presentable, if the large number of outstanding cases where dentistry has been the means of relieving serious disorders after the routine treatment of medicine had failed were conspicuously brought to the attention of the people so that our wealthy men could become familiar with them, there is no question that this science would be fostered by adequate aid.

Possibly one explanation of the fact that dentistry has failed to make a deeper impression on philanthropists is that it requires so much of the mechanical to dispense its service, and the consideration of mechanics does not appeal to public spirited men as does any kind of research. But dentistry is just as much a depart-

ment of health service as is any other branch of the healing art. This was fully recognized by such philanthropies as the Forsyth and Eastman endowments, whereby large sums of money have been donated for the care of children's teeth. If these splendid men had not known that attention to the teeth of children would contribute in a concrete way to the furtherance of the health of the coming generations, they would never have devoted so much money to establish dental infirmaries. All honor to the Forsyths and the Eastmans, the men who first caught the vision of the beneficence of this work. And all honor to Mr. Julius Rosenwald, who did so much in former days for the children of Chicago, and also to other splendid men who have contributed to dental relief.

What we now need is endowments from some of the great foundations to foster research and education in dentistry as is being done in medicine. The idea that dentistry will ever reap any benefit from endowments to medicine either in research or in education is wholly visionary. To achieve anything tangible for dentistry, the endowment must come directly to that profession, and not be relayed through any other source. The experiment of conducting a dental department in a medical school has always turned out an utter failure. Medical men do not think in dental terms any more than dentists think in medical terms. It is, of course, necessary that the two cooperate in the closest way in the treatment of disease, and that physicians and dentists consult with each other in the management of ailments affecting the teeth and jaws; but in the methods of educating students, the dentist would be no more wide of the mark in attempting to direct a medical school than would the physician in trying to direct a dental school.

With approximately 50,000 dentists serving the people of America, it becomes essential to have a great deal of research, to the end that this service be dispensed along intelligent lines. Think of the added good that would come to our people if all of our dentists possessed more light on the one subject of the prevention of dental decay. If scientific research could only illuminate a single problem of the many that confront dentists, it would at

once bring to our citizens benefits both tangible and concrete.

And if these problems are ever to be solved, it must be through the initiative of dentists themselves favored by sufficient funds to carry out the work. Dental research must be conducted by dentists, for dentistry, and to the ultimate end that the people are better served in a dental way. If this ideal is made fully effective, it will be only because our philanthropists or foundations make liberal endowments for the specific purpose of fostering dental education and research.

STYLES FOR NOVEMBER

In the November of life, there are certain styles that I should like to recommend. I wouldn't wear a coat of pessimism if I were you—leave that to the sophisticated youth of today, who assume to know more of life than did all the generations before them—and probably they do. Pessimism never fits a normal human being perfectly anyhow; it is never becoming, and doesn't hang well. Pessimism was intended for the select few, the few who were born to reform the world and didn't succeed. Pessimism is only another name for disappointment, and disappointment is never constructive in character.

An excellent garment for November is good cheer. Nothing tends to keep one feeling warm like that, and nothing is more becoming. It is an appropriate garment for the year round, and is never out of season. I recommend it with assurance, never having known this garment to be returned for lack of fit. It is not the thickness of the cloth either that makes the garment of cheer so warm—it is the character of the goods. It is perfect for November, just when the nipping frosts of autumn are growing severe, and something is required to keep out the chill.

Another fine kind of raiment is charity—something that is not always worn in November. This garment is beyond all praise, and the style doesn't change generation after generation. Charity and its handmaiden tolerance go to make up a really remarkable raiment for every individual, and the best part of it is that it may

be worn with comfort in any climate. It seems to keep out extreme heat and extreme cold, and it is suitable for every occasion.

In November, we often see a garment used that is not quite appropriate: a cloak of prejudice against the prevailing customs of the day, a constant harping on the virtues of the past and a failure to recognize anything good that was invented yesterday or today. The tendency to revile the present and assume that the social fabric of the days gone by was made of superior material that can never again be duplicated is accountable for much of the unhappiness that sometimes assails the autumn of life. Today has its faults; but compared with some of the past, today is millennium; and there has never been a better age than this. The garment of prejudice should never be worn in November—or any other time. Plant the seeds of hope and optimism, and the fiber that comes from a crop like that will clothe with joy the chilling hearts of men. All the virtues didn't die when the generations of the past ceased their activities, and all the vices have not been born since May became November.

Just because it is November, there is no reason why no provision should be made for the future. Too often when November comes, it is assumed that the end of the journey is near, and there is no need of reckoning with the time to come. Not only is this attitude illogical but it also invariably results in shortening the time; and who wants to do that if he is normal and not suffering? Make plans for the future and take it for granted that you will need garments for a long time yet. Even if you do die soon, the garment may come in handy for a shroud: it will not be wasted. Don't assume that the work is all done yet. It will never be done; and the more you do, the more you will want to do and the sweeter life will be. Keep busy. Your judgment may be more useful than that of those who have had less experience. Don't lose your nerve.

One of the most fitting garments for November is laughter. There is never an age when this is not needed, and the more it is worn, the better. Laughter is the music of happiness, the song of the celestial spheres, the pean of the human heart. It vibrates on the ambient air, and ripples like wave lengths over the minds and

moods of men. It changes a dreary day into the sunshine of heaven's own blue, and casts its beams into every happy home. Laughter is the tonic of the world. It livens youth, glorifies the days of middle life, revives the drooping spirit of old age. Laughter is one of man's greatest blessings, and his most benign benefactor. Let every one laugh.

The running mate of laughter is humor, and the bright hued garment of humor should be worn as one of the most approved styles for November. Humor turns a tragedy into a comedy, a tempest into a gentle zephyr. Humor takes the somber out of life and substitutes for it the blessed balm of mirth. Where humor reigns, there dies the blighting plague of prudery and pretence; and in the human heart, there is no saving grace like this.

The knack of wearing garments is an art, and in November days, when vision dims a bit, there comes the need for greater care, and trimmer, neater modes. The cycle of the year brings summer, winter, spring, and while November holds the name of being sad, styles may clothe the autumn days with warmth and light and grace. It may also bring, depending on our mood, a wealth of beauty, hope and cheer.

To be beautiful in November is a blessing; to be hopeful at any time is a privilege, and to be cheerful when the leaves are fluttering toward the earth is the sweetest solace that can touch the heart of man.

THERAPEUTICALLY SUGGESTIVE NAMES AND THE LAITY

Elsewhere in this issue of THE JOURNAL appear several reports on dentifrices of the patent-medicine type. In each case, report is made on preparations sold under therapeutically suggestive names which in no way indicate the composition of the product. The consumer, be he dentist or layman, if left in the dark concerning it. For long years, mystery and secrecy have been the handmaidens of manufacturers who promote unscientific remedies and dentifrices. This illustrates but one phase of the important

work facing the profession, which is under way as part of the A. D. A. Chemist's program.

Therapeutically suggestive names have long been condemned by practitioners who endeavor to hold high the standards of their calling. They realize that the danger lies not only in the possibility of injurious ingredients in a preparation, but also in the irresponsible claims made for such products by their promoters and the indiscriminate use to which such products are put by the laity. In the case of "Tartaroff" (and others of like character), the name suggests even to the most ignorant layman a preparation for the removal of tartar; in which case a little knowledge is worse than none. In such cases, the user may permit himself to be led astray by the extravagant claims made by the vendors of these products with consequent loss of tooth-function, instead of seeking the services of the profession most qualified to give proper treatment. Furthermore, of equal importance is the fact that the profession must stand foursquare against the use of meaningless names for shot-gun mixtures or preparations, as will be taken up in a future editorial.

Brief mention is made of preparations sent out under the fanciful names of "Knox-Tartar" and "Miracle Pyorrhea Powder," which, while not containing particularly harmful ingredients, may be potent agents for harm. For the misguided user of such preparations is led to rely on the extravagant claims accompanying a catchy name instead of securing proper treatment for a condition which may indicate more serious underlying conditions.

The use of therapeutically suggestive names is not consistent with the ideals of an advancing and public-serving profession. The profession will do well to insist on the eradication of deleterious products exploited either through the dentists or directly to the laity under names suggestive of therapeutic conditions in which they are to be used; products which are secret in composition. The profession alone, not vendors of worthless preparations, is qualified to diagnose and prescribe.

DEATHS UNDER ANESTHETICS

The Dental Surgeon of London, England, is looking up data regarding deaths under anesthetics, and is desirous of getting particulars connected with such deaths from members of the profession in all parts of the world. We trust that our readers will cooperate in every way possible, and that, in the event any accident of this kind has come under their observation, they will forward every possible particular to the publishers, Baillaire, Tindall & Co., 8 Henrietta St., Covent Garden, London, England.

JOHN A. STOECKLEY, D. D. S.
(1870-1928)

By R. N. DOUGLAS

John A. Stoeckley, of South Bend, Ind., one of the leading members of the dental profession in his state, died suddenly in his home, 609 Portage Avenue, September 24. Dr. Stoeckley had not been well for the past year, but lately was much encouraged by an improvement in his health. He spent the day of his death in his office.

He was a native of Ohio, where he was born in Monroeville, May 20, 1870. He studied pharmacy at Western Reserve University, Cleveland, Ohio, and continued in that profession until 1893, when he entered the Chicago College of Dental Surgery, from which he graduated with honor in 1896. The same year, he located in South Bend and opened an office, practicing there continuously for thirty-two years.

Dr. Stoeckley was a member of the surgeon's staff of St. Joseph's Hospital, South Bend. He always manifested a keen interest in dental society affairs, being actively associated with various dental organizations during practically his entire professional career. He was a life member and held various positions of responsibility in the Indiana Dental Society. He was a past president of the Northern Indiana, Thirteenth District and St. Joseph County dental societies, and he was also identified with the American Dental Association, the American Society of Oral Surgeons and Exodontists, and the National Anesthesia and Research Society. He was a member of the Delta Sigma Delta Fraternity.

Dr. Stoeckley was married to Miss Emelyn Hinkle, of Muncie, Ind., June 18, 1902. Three children were born to this union, John, Hildegard and Marion, who, together with the widow, survive him.

BUREAU OF CHEMISTRY

TARTAROFF

A "Tartar-Remover"

TARTAROFF is a preparation sold by the Tartaroff Company, Chicago, Ill. Some of the claims made for the product are:

Marvelous Discovery for the Teeth. Makes Them as White as Snow. Tartaroff acts like magic on the teeth.

It instantly removes all film, tartar, stains and spots caused by tobacco, nicotine, fruits, vegetables, medicines or ugly discolorations.

It also says: "Tartaroff makes old, yellow, ugly teeth glisten like polished ivory."

Other claims are:

Tartaroff is the greatest scientific discovery of the age. Nothing like it ever prepared before. It is not a tooth paste but a simple, harmless preparation that can be applied to the teeth in a few seconds. Immediately the teeth are transformed into gems of pearl white beauty. Tartaroff will not injure the most delicate gums, enamel, gold, silver or other fillings.

How often have dentists heard similar remarks!

In order that the public and the dental profession might know something about this "greatest scientific discovery of the age" for the removal of tartar and other agents, the A. D. A. Chemist was asked to investigate the product. The Chemist's report follows.

REPORT OF THE A. D. A. CHEMIST[1]

"Six original packages of 'Tartaroff' (Tartaroff Company, 1306 and 1308 West 74th Street, Chicago, Ill.), price 25 cents per bottle, were purchased on the open market for examination.

"The following appeared on the label:

TARTAROFF

Makes your Teeth Pearly White in the magic of a moment. Tartaroff is guaranteed to remove Tartar, Film, Nicotine and all ugly stains from your Teeth. Dampen Tooth Brush or Cotton Swab with Tartaroff Freely, Rub Teeth Briskly, then use Tooth Paste as usual.

"Each original bottle contained 7.5 c.c. of a clear, red, odorless liquid, possessing a sour taste. The contents of six bottles were mixed and used for the chemical investigation.

"The liquid was acid in reaction toward litmus; its p_H value was found to be 1.0.[2] The specific gravity was 1.006.

"Qualitative tests indicated the presence of hydrochloric acid, and a minute trace of aluminum. Fluorids, phosphates, sulphates, oxalates, nitrates and other cations (metals) were not found.

1. The details of analysis will be published elsewhere.

2. As will be recalled, p_H is the modern physicochemical symbol used to express the degree of acidity and alkalinity. For instance, water, which is neutral, has a value $p_H = 7$. All values numerically below 7 indicate relative acidity; thus, the p_H of gastric juice—that is, 0.2 per cent hydrochloric acid—is 1.7. The p_H of the normal saliva is approximately 6.8 to 7, while that of blood is 7.4-7.6.

"Quantitative determinations yielded the following:

Total solids	0.68 per cent
Ash	0.008 per cent
Hydrochloric acid	1.23 per cent

"From the foregoing, it may be concluded that a product having the essential composition of Tartaroff may be made by adding 3.5 c.c. of hydrochloric acid, U.S.P., to 100 c.c. of water and adding a small amount of methyl orange to the solution to give the desired red color."

In the light of the Chemist's report, to state that the claims made for Tartaroff are exaggerated would be putting it very mildly. As is shown in the report, the product is essentially a solution of hydrochloric acid sold under a noninforming name—a name which may easily lead to indiscriminate use of the preparation in all "tartar conditions" of the mouth. Furthermore, the name is therapeutically suggestive and undoubtedly may be the means of harmful self-treatment by the laity, in conditions which may require the best of dental care.

Thus, stripped of its secrecy and the concomitant mystery, the "Greatest Scientific Discovery of the Age" stands revealed as nothing more than ordinary hydrochloric acid, diluted with water and colored, for sale under a fanciful name to an unsuspecting and uninformed laity at an outrageously exorbitant price.[3] If the claims of the Tartaroff people were given credence, the analysis would indicate that scientific discovery has not emerged from the dark ages.

The claim that 1.2 per cent hydrochloric acid solution is harmless can come only from one who is uninformed as to the destructive action of it on the enamel and other parts of the tooth. Removal of tartar and other concretions on the teeth should be left to the care of dentists. "Tartar-conditions" cover a multitude of pathologic conditions, many of which indicate more serious underlying conditions.

Thus, such preparations may be potent agents for added harm by giving a sense of false security. If such a dangerous fluid as hydrochloric acid is indicated, it must be used with extreme care on the teeth, and then only in the hands of a skilled dental operator.[4] It should never be used by the laity. The profession would do well to inform the laity of the dangerous character of this preparation, and the injurious result of its use.[5]

Other Dental Preparations

From time to time, inquiries regarding the products which are within the purview of the A. D. A. Chemist are made. Owing to the fact that analyses or investigations of some of these products have been published elsewhere, it is deemed best to refer to previous articles. The following are typical:

"Knox-Tartar"[6]

A correspondent requests information concerning the composition of "Knox-Tartar."

According to a recent article, "Knox-Tartar" (A. W. Knox, Los Angeles) is a preparation for cleansing the teeth. The carton declares that the product contains "no acid" and "no pumice." Microscopic and chemical examination

3. The cost of the ingredients of each bottle of Tartaroff may be calculated to be much less than a penny.

4. For analyses of similar dangerous preparations, the reader is referred to the exposé of "Taxi" and "Stain-Remover," J. Dent. Res., 7:477, 1927.

5. The New Hampshire State Board of Health has pointed out the dangers of Tartaroff (Health, January, 1928).

6. J.A.M.A., 89:2059, 1927.

Marvelous Discovery for the Teeth
Makes Them White as Snow
TARTAROFF acts like magic on the teeth. It instantly removes all film, tartar, stains and spots caused by tobacco, nicotine, fruits, vegetables, medicines or other ugly discolorations. Makes old, yellow, ugly teeth glisten like polished ivory.
TARTAROFF is the greatest scientific discovery of the age. Nothing like it ever prepared before. It is not a tooth paste but a simple, harmless preparation that can be applied to the teeth in a few seconds. Immediately the teeth are transformed into gems of pearl white beauty. TARTAROFF will not injure the most delicate gums, enamel, gold, silver or other fillings.
Get a bottle today at your local drug store. Enough for two months, only 25c. Try it and if not more than pleased with the results the druggist will return your money.

Reproduction of advertisement for Tartaroff.

indicated the presence of a diatomaceous earth (*Kieselguhr*), starch and silica (sand). The article concludes thus: "Knox-Tartar appears to be essentially an abrasive (*Kieselguhr*) to which has been added a very small amount of starch."

Charles F. Bödecker states[7]: "Two kinds of ingredients sometimes found in dentifrices may do considerable harm to the enamel: a. The acid group; b. The gritty group." *Kieselguhr* belongs to the latter group.

"Miracle Pyorrhea Powder"

Some of the claims made for "Miracle Pyorrhea Powder" (Miracle Remedy Co., Detroit, Mich.) are:

> Let us save your teeth! No matter how soft or how spongy and bleeding the gums may be, or the teeth so loose it seems you could pick them out with the finger, the Miracle Pyorrhea Powder will make them hard and firm again.

The Toledo Better Business Bureau sought information regarding this "Miracle." The laboratory from which this report is taken states: "It appears from the chemical analysis that Miracle Pyorrhea Powder is essentially a mixture of baking-soda and borax, to which has been added a very small amount of aromatic oil."

Further comment seems unnecessary.

7. J. Dent. Res., 8:426, 1928.

BUREAU OF DENTAL HEALTH EDUCATION

THE INCIDENCE OF DENTAL DEFECTS IN TWO THOUSAND FOUR HUNDRED AND SIXTY-NINE EMPLOYMENT EXAMINATIONS*

By C. O. SAPPINGTON, M.D., Dr. P.H.,† Oakland, Calif.

RECENT developments in the field of industrial health have laid emphasis on the value of industrial dentistry. Most authorities have agreed to the proposition that, in order to provide properly for the health of the worker, medical and dental services must supplement each other. This conception has assumed importance from several standpoints: (1) relative to the aggravation of injuries or the delay of healing and repair of sprains, strains, contusions, fractures or wounds, because of the existence of focal infection in the teeth, and (2) as a means of preventing the group of ailments which may arise as a result of dental and periodontal disease. These conclusions have been arrived at through statistical studies, which have shown that focal infection and its effects have been charged with being the chief cause of inefficiency, absenteeism, sickness and prolongation of convalescence after injuries, in the history of American industrial and mercantile medical experience.

†Medical director, Montgomery Ward and Company, Oakland, Calif.

*Read before the Fifty-Eighth Annual Meeting of the California State Dental Association, Stockton, Calif., April 9, 1928.

SOURCE OF MATERIAL IN THIS STUDY

While it is duly recognized that the chief consideration in industrial dental practice is the correction of abnormal conditions as found, the value of the enumeration, classification and study of various concomitant factors affecting observed dental defects is also of prime importance; for it is only through compilation and comparison of such experiences that we shall finally be able to say what is very common, what is average and what is rarely encountered.

With these ideas in mind, the dental defects found during 2,469 employment medical examinations at the Oakland plant of Montgomery Ward and Company have been collected, tabulated and studied.

DEFINITION AND CLASSIFICATION OF DENTAL DEFECTS

Necessarily, one must first define what is meant by a dental defect, and, secondly, he must attempt to classify the different kinds of abnormalities found in this series of examinations.

We have taken as our definition of a defective dental condition any demonstrable change in the structure of the tooth or pericemental tissues that may be

construed as being a deviation from the normal. (An exception is made in the exclusion of orthodontic defects from this series, chiefly because of the difficulties of recognition of the existence of such changes, without the use of time-consuming procedures and also because of the complicated arrangement resulting after attempts to classify degrees of malocclusion. For the same reasons, no account has been taken of the occurrence of devitalized teeth.)

We have used, throughout this study, the following simple outline for dental defects:

1. Restorations (any filling).
   - (a) Small—from pinpoint size to about a diameter of 3 mm.
   - (b) Moderate size—from 3 mm. diameter to half the surface restored (mesial, distal, lingual, etc.).
   - (c) Large—over half of surface restored.
2. Caries (A visible carious process).
   - 1°—As in small restorations.
   - 2°—As in moderate size restorations.
   - 3°—As in large restorations.
3. Restored teeth (all artificial teeth).
4. Missing teeth.
5. Crowned teeth.
6. Pyorrhea.
   - Incipient (Any degree of gingivitis).
   - Moderately advanced (Soft spongy gingivae which bleed easily on pressure and show definite retraction.)
   - Advanced—much retraction—pus.

Incidental to this study, a grading scheme has been invented. By subtracting from a perfect score the amounts assigned to each defect as above noted, it is possible to grade an individual in terms of his or her gross dental abnormalities. Analysis of this part of the work will be made in a separate paper.

COMMENTS ON THE DATA

By reference to Tables 1 and 2, it will be seen that (1) 55.2 per cent of the group were under age 20; (2) 89.7 per cent of the group were under age 30; (3) 55 per cent were males; (4) 45 per cent were females.

Table 3 shows the incidence of defects in the order of their frequency of occurrence.

In Table 4, the following points have been observed: 1. Restorations and missing teeth accounted for 84 per cent of the total number of defects noted; pyorrhea made up 0.3 per cent of the abnormal conditions. 2. Females showed a lower percentage of caries and a higher percentage of restorations and missing teeth than males. 3. The percentage occurrence of all defects was evenly divided between males and females.

An interesting point was observed in Table 5, i. e., the incidence of the average number of defects per person steadily increased in the age divisions given up to group 40-49, where it was highest. All groups were above the general average except that of "under age 20," which was below the general average.

Tables 6 and 7 show that the highest percentages of defects were found in the lower age groups.

It was thought well to compare the percentage incidence of defects with the percentage incidence of persons by age group and sex. This was done in Table 8, with the result that in all age groups except "under age 20" the percentage of defects was higher than the percentage of persons in the same group.

Table 9 should be of interest as it has shown that the total average percentage of missing teeth restored was only 4.9. In general, the highest percentages occurred in the groups 20-29 and 30-39; whereas, the lowest occurred under age 20.

In Table 10, we have taken the number of all fillings observed and added this number to the number of all the carious processes seen. Using this numerical result as a basis, we have computed the percentage of possible carious teeth which have been filled, by reference to the actual number of carious defects seen. The results were: 1. Females showed a higher percentage throughout than males, group for group. 2. In general, the divisions 20-29 and 30-39 showed the highest percentages.

SUMMARY

The incidence of the common types of dental abnormalities have been given, the source of the material being employment, examinations in a mercantile organization, 55.2 per cent of the subjects being under age 20. The following points were established in this study:

1. Restorations, fillings and missing teeth accounted for 84 per cent of the deviations from a normal state.

2. In general, the abnormalities observed were divided fairly evenly between males and females.

3. Apparently, females took better care of the teeth as they showed a lower percentage of caries and a higher percentage of restorations (fillings).

4. The incidence of the average number of defects per person increased directly with the age up to the 40-49 group (which was the highest). The only group below the total average was that division of "under age 20."

5. Counting all defects, the highest percentages of defects occurred in the lower age divisions (probably because of the age distribution). On the other hand, the percentage of defects was greater than the percentage of persons in all groups except that of "under age 20."

6. Only 4.9 per cent of missing teeth were found to be restored.

7. Of all possible carious teeth, 88.2 per cent were observed to be filled. Females average higher than males in this regard throughout all age segregations. The highest percentages consistently occurred in the groups 20-29 and 30-39.

312 Wayne Avenue.

TABLE 1.—AGE AND SEX GROUPING

	Under 20	20-29	30-39	40-49	50-59	60+	Total
Males	769	430	87	49	22	3	1,360
Females	597	422	72	17	1	0	1,109
All persons	1,366	852	159	66	23	3	2,469

TABLE 2.—AGE AND SEX GROUPING (BY PERCENTAGE)

	Under 20	20-29	30-39	40-49	50-59	60+	Total
Males	56.6	31.6	6.4	3.6	1.6	0.2	55.0
Females	53.8	38.1	6.5	1.5	0.1	0.0	45.0
All persons	55.2	34.5	6.5	2.7	0.9	0.2	100.00

TABLE 3.—OCCURRENCE OF DEFECTS (BY SEX)

	Males	Females	All persons
Restorations	4,822	4,837	9,659
Missing teeth	1,643	1,705	3,348
Caries	793	497	1,290
Crowned teeth	518	474	992
Teeth restored	80	83	163
Pyorrhea	32	7	39
Total defects	7,888	7,603	15,491

TABLE 4.—OCCURRENCE OF DEFECTS (BY PERCENTAGE, ACCORDING TO SEX)

	Actual Total Number	Defects, Male Per Cent	Defects, Female Per Cent	Total Defects Per Cent
Restorations	9,659	61.2	63.7	62.4
Missing teeth	3,348	20.6	22.4	21.6
Caries	1,290	10.1	6.5	8.3
Crowned teeth	992	6.6	6.2	6.4
Teeth restored	163	1.0	1.1	1.0
Pyorrhea	39	0.5	0.1	0.3
Total defects	15,491	50.9	49.1	100.0

TABLE 5.—AVERAGE NUMBER OF DEFECTS PER PERSON IN AGE GROUP AS NOTED

	Under 20	20-29	30-39	40-49	50-59	60+	Total
Males							
Number in group	769	430	87	49	22	3	1,360
Average number defects	5.0	6.3	7.1	10.2	6.6	9.3	6.1
Females							
Number in group	597	422	72	17	1	0	1,109
Average number defects	5.5	7.7	12.1	12.2	36.0	0.0	6.9
All Persons							
Number in group	1,366	852	159	66	23	3	2,469
Average number defects	5.1	7.0	9.3	10.7	7.9	9.3	6.3

TABLE 6.—OCCURRENCE OF DEFECTS (BY AGE GROUP AND SEX)

	Under 20	20-29	30-39	40-49	50-59	60+	Total
Males	3,867	2,725	620	502	146	28	7,888
Females	3,261	3,232	867	207	36	0	7,603
All persons	7,128	5,957	1,487	709	182	28	15,491

TABLE 7.—PERCENTAGE OF DEFECTS (BY AGE GROUP AND SEX

	Under 20	20-29	30-39	40-49	50-59	60+	Total
Males	49.0	34.6	7.8	6.4	1.8	0.4	50.9
Females	42.8	42.5	11.4	2.7	0.4	—	49.1
All persons	46.0	38.5	9.6	4.6	1.2	0.1	100.0

TABLE 8.—COMPARISON OF PERCENTAGE OF DEFECTS WITH PERCENTAGE OF PERSONS (BY AGE GROUP AND SEX)

	Under 20	20-29	30-39	40-49	50-59	60+	Total
Males							
Defects	49.0	34.6	7.8	6.4	1.8	0.4	50.9
Persons	56.6	31.6	6.4	3.6	1.6	0.2	55.0
Females							
Defects	42.8	42.5	11.4	2.7	0.4	—	49.1
Persons	53.8	38.1	6.5	1.5	0.1	0.0	45.0
Both sexes							
Defects	46.0	38.5	9.6	4.6	1.2	0.1	100.0
Persons	55.2	34.5	6.5	2.7	0.9	0.2	100.0

TABLE 9.—PERCENTAGE OF MISSING TEETH RESTORED

	Under 20	20-29	30-39	40-49	50-59	60+	Total
Males	3.2	5.7	7.4	4.5	8.0	0.0	4.9
Females	5.1	5.9	4.7	0.0	0.0	—	4.9
All persons	4.0	5.7	5.6	3.2	5.1	0.0	4.9

TABLE 10.—PERCENTAGE OF CARIOUS TEETH FILLED

	Under 20	20-29	30-39	40-49	50-59	60+	Total
Males	84.8	90.2	88.3	71.2	87.3	0.0	85.8
Females	87.8	93.2	95.0	100.0	0.0	0.0	90.7
All persons	85.8	91.6	92.1	79.9	87.3	0.0	88.2

CHILDREN'S DENTISTRY FROM A PUBLIC HEALTH, AS WELL AS THE PRIVATE PRACTITIONER'S STANDPOINT

By L. W. MORREY, D.D.S., Chicago, Ill.

SO MUCH has been written in the past few years concerning children's dentistry, or the general care of children's teeth, that we are forced to the conclusion that the time will soon be at hand when this most important branch of dentistry will receive the same recognition that other branches of our profession now receive. You will note that I say "the time will soon be at hand." It is not yet here. As much as it is talked, preached and glorified, children's dentistry is not practiced by the profession, nor is it now being given the honest attention of the general dental practitioner that it deserves.

Looking at this particular department of dentistry with the aid of rose colored glasses, we find several good and simple reasons why children are not receiving the same dental attention that we, as a profession, are endeavoring to give our adult patients. If we will stop to consider that, after all, dentists are merely human, we will see how perfectly simple

it is to explain why the care of children's teeth is being neglected.

It is only natural that each of us should develop into or gradually specialize in that branch of dentistry which, to us, is the most agreeable, and the most profitable and also is that branch in which we honestly feel that we can give the best service. For instance, those of us who prefer the mechanical side of dentistry sooner or later specialize in crowns and bridgework; others, who are proficient in removing teeth, soon either specialize entirely or in part in exodontia; still others become interested in pyorrhea and devote their energy and time to that specialty. A few specialize in oral surgery. Almost any community of any size has its denture specialist. But how many men in the profession can you name who are specializing in children's dentistry?

What do those facts mean? Simply this: A dentist, being human, is not going to confine his practice to the care of children when he knows that he can give better service to the general public by confining himself to some other branch of dentistry. Being human, few dentists are going to submit themselves to the mental and nervous strain of handling recalcitrant children all day long when they can devote the same amount of time and expend one-fiftieth the amount of nervous energy to making full dentures. Furthermore, at present, few dentists get the same fee per hour for children's work that they do for adult work. There is no use arguing that we should get it; I know that we should get it, but we don't. That day will come; but this paper deals with the present day conditions as I see them.

These facts being true, it is quite evident that no dentist with a full and active practice will devote all or even a large part of his time to children's dentistry when he can devote the same time more lucratively to the care of adult patients. Add to this the fact that our living conditions such as diet, housing, exercise, nervous expenditure and rest periods have materially changed in the past three or four generations, and that the general public, as well as a large majority of the dental profession, have been slow to recognize their deteriorating effect on children's teeth, and we have four good reasons why from 85 to 90 per cent of our children are suffering from diseased teeth and their subsequent systemic disorders.

Let us try to look at the situation clearly. We have, on the one hand, from 85 to 90 per cent of our children with decayed teeth. We all know that each one of those decayed teeth is a menace or may become a menace to the child's health and mentality. We, as members of the dental profession, know that it is our duty and our duty alone to correct these conditions. On the other hand, after studying the situation in various parts of the State of Illinois, I am forced to the conclusion that we are far from adequately meeting the situation as we should. We have not assumed to the full our great responsibility in guarding the health of our community.

To combat dental decay successfully and completely we, as a profession, must attack this serious problem from two distinct angles, just as the medical profession is attacking its problems from two angles. 1. Each of us must make a more thorough and intelligent and determined effort to care for children's teeth in our private practice. 2. We must take advantage of public health agencies and utilize them as our agents to educate the public in matters of mouth

their confidence, there is nothing he cannot do for them. Quite naturally, the first step to be taken is to relieve pain; next, the minor operations should be undertaken. After the third or fourth sitting, when a complete understanding exists between the patient and the operator, almost any operation, even though most painful, can be accomplished without fear of breaking that confidence.

Never allow a child to leave your office with a feeling of resentment toward you. If an operation has been exceedingly painful, find a few minutes of your time to get back in the good graces of the patient. Make the patient see that you dislike hurting him, but that it was necessary. By assuming the proper mental attitude toward your child patients, you can, in a short time persuade most of them to consent to your operations. They may not like it, but if they have confidence in your truthfulness and ability, they will steel themselves to almost any pain or discomfort.

So much has been written about the importance of early care of the first permanent molar, especially the filling of the grooves and fissures, that we will not go into that at this time. I must, however, speak of the care of deciduous molars, especially the second deciduous molars. Thousands of these little teeth are becoming decayed, and even though they may be filled, they, in turn, are causing decay on the mesial aspect of the first permanent molars shortly after those teeth erupt.

It is most important that those second deciduous molars receive early and competent dental attention. Quite often, these teeth are filled with cement or with amalgam, and no attention is paid to the contour or contact of the filling. If this amalgam filling is placed on the distal surface of the second deciduous molar, and no thought is given to the contour, food will lodge between it and the first permanent molar when that tooth erupts. In a short time, the mesial wall of the first permanent molar will be etched through, and decay sets in. This is especially true when cement fillings are used as filling material in distal cavities of second deciduous molars. The cement soon washes away, the contact is destroyed and the first permanent molar is soon in need of a mesiocensal filling. I cannot urge too strongly that more care be used in filling second deciduous molars. The type of filling material used and the contour of the filling must be of the highest quality, not only for the sake of the deciduous tooth, but for the sake of the mesial surface of the proximate, or soon to be proximate, first permanent molar.

Quite often, in preparing a cavity in a second deciduous molar, it becomes apparent that to remove all the decay fully, the nerve will become involved. Any attempts that I have ever made to sterilize the remaining leathery dentin and insert a filling over it have resulted in disaster; therefore, I always make it a practice to remove all the decay before filling. If, as I say, I see that the pulp will become involved in so doing, I seal in a very minute particle of devitalizing paste together with a dressing of desensitizing liquid, with some good dependable cement. This should be left in for twenty-four hours. At the second sitting, the coronal portion of the pulp will be easily removed, and a dry dressing of formocresol is placed in the pulp chamber. This is allowed to remain twenty-four hours. At the third sitting, a paste of formocresol and thymolized calcium phosphate is placed in the floor of the pulp chamber, over which a base of cement is laid. A copper band which

has been festooned and trimmed to fit the tooth closely is then placed over the crown, and an amalgam filling inserted. Because children are liable to distort an amalgam filling before it is sufficiently set, I like to use a copper band for a matrix. This can be removed a few hours later, or can be allowed to remain until the next day and then be removed. By treating badly decayed deciduous teeth in this manner, I have had fewer failures, fewer abscessed teeth, with less pain to the little patient and fewer disgruntled parents than with any other method of procedure. It is the most direct, simple and efficient method that I know of to employ in treating such cases, and a deciduous tooth so treated will give good service for several years.

As you know, it is most important that we maintain the space occupied by the second deciduous molar. Quite often, there are cavities on both the mesial and the distal surfaces of these teeth. If, in removing the decay, I find that so much of the tooth is involved that the remaining structure is too weak to hold an amalgam filling, we proceed as before. At the first sitting, I devitalize the tooth; at the second, I treat it with a dry dressing of formocresol, and at the third, I seal in a dressing of thymolized calphosphate and formocresol under the amalgam filling. After the amalgam has set, I shape the buccal and lingual surfaces and the occlusal surface and place a small gold shell crown over the entire restoration. Thus, the space will not only be retained mesiodistally but gingivoocclusally as well. If care is taken in shaping the crown, perfect contact can be established with the first permanent molar and no interproximal decay will result.

Under no condition do I think it advisable to so treat abscessed deciduous teeth. I much prefer to extract them, as I consider that procedure the lesser of two evils. I might also mention that the general systemic condition of the child regulates to a greater or lesser degree how many devitalized deciduous teeth I leave in the mouth.

SUMMARY

May I say that our state society, together with other societies, believes that we, as an organization, have a bigger and broader field of endeavor than that which is confined within the four walls of our offices. If we honestly wish to combat disease; if we as professional men and women want to live up to the high ideals of our profession, we must use our united influence in educating the public as to how they themselves can develop good teeth and thus stamp out this ever-growing spread of dental decay, in the wake of which follows a host of general systemic disorders. If each component society will seriously and energetically undertake the task of educating and enlightening the community on the subject of mouth hygiene; if each component society will appoint a committee that will enthusiastically carry out the dental phase of a community health program; if each component society will take the initiative in health matters in its district, and will see to it that some of its members act on school boards or in other lay groups which are interested in community affairs; then, if each of us will individually give to each of our host of child patients the care and attention that we are capable of giving and the kind of service that we, ourselves, would like to receive, it will not be long until the dental profession in the State of Illinois will be taking its rightful place as a leader in health work rather than as a servant at the beck and call of uninformed lay organizations.

DENTAL HYGIENE AS TAUGHT IN THE SCHOOLS OF WATERLOO, IOWA*

By CLELLA McCULLOUGH, Waterloo, Iowa

WE HAVE gradually been placing the hygienist in the public schools over the State of Iowa. Four years ago, Waterloo placed herself third on the list of cities in Iowa which have established a system of mouth hygiene in the public schools. Des Moines is first, having started this work eleven years ago. Sioux City is next; and, this September, Dubuque places herself fourth on the list. This year, one hygienist is employed by the Extension Division of the University of Iowa, and will carry out a plan for dental service throughout the city, town and country schools wherever her service is desired.

This plan has been tested in Waterloo and has proved to be very successful. A four years' trial has developed a real interest in the proper care of children's teeth.

The hundreds of eager eyes and smiling faces into which I have looked the past few years will be my inspiration as I tell you of our work. It is to make more eyes bright and more faces smiling that we want to tell you what we have been able to do in the public schools of Waterloo and what can be done in preventive dentistry everywhere. To not only the boys and girls who are in school now but to the never-ending procession of children who are to enter our educational institutions during the coming years, we want to bequeath a sound, healthy body, clean habits, clear mentality, sound morality and happiness. Mouths free from disease will aid in this work.

Our task is to impart dental truths to the children in such a way as to stimulate an interest about his teeth in the child himself and, through the child, to develop the interest of the parents and encourage the parents to care for the child's teeth.

For an example, when the teeth of one boy were examined for the first time, he was told that there were cavities in all his permanent molars and that they must be filled in order to save the teeth and that he must start using his toothbrush three times a day. This was his first conception of the dental hygienist's work and his awakening to what this new kind of a teacher was trying to do for him. When Johnny found that he could be a bigger and better man by taking care of his mouth, mother and father were glad to take him to the dentist, if only to escape further annoyance. But some of the children whose interest had been fanned into an earnest desire to have a clean mouth were so unfortunate as to have parents who would not cooperate. This attitude of their parents would have discouraged a grownup and dampened their desire for good teeth.

*Read before the Section on Mouth Hygiene and Preventive Dentistry at the Seventieth Annual Session of the American Dental Association, Minneapolis, Minn., Aug. 21, 1928.

But the buoyancy, persistence and ardor of children is the saving grace which enables the hygienist to carry out her campaign for better teeth. Once a mass movement is started, nothing will stop the ardor of these children. Whether it is bees, birds, butterflies or bugs, get a class of children enthused, and you will have results that last a life time even though the noise of enthusiasm for the subject wanes. So by applying this psychology, we have worked out what we call "The Iowa Plan for Dental Service," with excellent results, as you will agree when the facts and figures show that, our first year, we had 56 per cent of sound teeth and the fourth year shows a gain of 78 per cent. The children are interested. As many as could had all the cavities filled and other necessary work done. The question to be answered is: "How can we keep them interested?" for they must have continued stimulus until tooth care becomes a habit.

In Waterloo, "The Iowa Plan for Dental Service" and the organization of the "Tooth Brush Club" answered this question.

The Iowa Plan for Dental Service has been carefully worked out by the Oral Hygiene Committee of the Iowa State Dental Society. It has been used successfully in city, town and country schools all over Iowa. According to this plan, the dentists do not enter the school, but they do the part of the work that no one but a skilled dentist can do. They remain in their offices and fill the teeth of hundreds of pupils sent to them every year by the dental hygienists, principals and teachers. There are five distinct parts to the program:

1. Examination of the teeth of each child by the dental hygienist with the mouth mirror and explorer. This examination upsets the school curriculum very little and is made with little loss of time. Any evidences of decay, stain, irregularity of the teeth or inflammation or swelling of the gums is sufficient cause for sending the child to his dentist for closer examination and attention. Such instructions for further attention are contained on the "blue notification card" that is given the child, which he is instructed to take home to his parents and then to his dentist. When the necessary work is completed, the dentist signs the card and it is returned to the teacher by the child. This card, properly signed and returned to the teacher, shows that the child has had the necessary dental attention, and makes it possible for the teacher to keep a complete dental record of all her pupils. The teacher keeps the record on a chart especially prepared for that purpose, called the "Room Record Chart." It is used by the hygienist at the time the examination is made to record the condition of each child's mouth and thereafter to credit the child with having the needed work done when he returns his card signed by the dentist. This chart gives, at a glance, a complete picture of the condition of the mouth of the child. Another chart, called the "Dental Honor Roll," is used to record the names of those who have brought their cards from the dentist properly signed It also serves as a means of checking the progress made in getting the pupils to obtain necesary dental attention. The effect on the pupils is twofold. Those who have not their names on the honor roll will do all that they can to get it there; those who have their names on the honor roll will do everything they can to induce the lagging ones to get their names on also.

DENTAL HYGIENE AS TAUGHT IN THE SCHOOLS OF WATERLOO, IOWA*

By CLELLA McCULLOUGH, Waterloo, Iowa

WE HAVE gradually been placing the hygienist in the public schools over the State of Iowa. Four years ago, Waterloo placed herself third on the list of cities in Iowa which have established a system of mouth hygiene in the public schools. Des Moines is first, having started this work eleven years ago. Sioux City is next; and, this September, Dubuque places herself fourth on the list. This year, one hygienist is employed by the Extension Division of the University of Iowa, and will carry out a plan for dental service throughout the city, town and country schools wherever her service is desired.

This plan has been tested in Waterloo and has proved to be very successful. A four years' trial has developed a real interest in the proper care of children's teeth.

The hundreds of eager eyes and smiling faces into which I have looked the past few years will be my inspiration as I tell you of our work. It is to make more eyes bright and more faces smiling that we want to tell you what we have been able to do in the public schools of Waterloo and what can be done in preventive dentistry everywhere. To not only the boys and girls who are in school now but to the never-ending procession of children who are to enter our educational institutions during the coming years, we want to bequeath a sound, healthy body, clean habits, clear mentality, sound morality and happiness. Mouths free from disease will aid in this work.

Our task is to impart dental truths to the children in such a way as to stimulate an interest about his teeth in the child himself and, through the child, to develop the interest of the parents and encourage the parents to care for the child's teeth.

For an example, when the teeth of one boy were examined for the first time, he was told that there were cavities in all his permanent molars and that they must be filled in order to save the teeth and that he must start using his toothbrush three times a day. This was his first conception of the dental hygienist's work and his awakening to what this new kind of a teacher was trying to do for him. When Johnny found that he could be a bigger and better man by taking care of his mouth, mother and father were glad to take him to the dentist, if only to escape further annoyance. But some of the children whose interest had been fanned into an earnest desire to have a clean mouth were so unfortunate as to have parents who would not cooperate. This attitude of their parents would have discouraged a grownup and dampened their desire for good teeth.

*Read before the Section on Mouth Hygiene and Preventive Dentistry at the Seventieth Annual Session of the American Dental Association, Minneapolis, Minn., Aug. 21, 1928.

But the buoyancy, persistence and ardor of children is the saving grace which enables the hygienist to carry out her campaign for better teeth. Once a mass movement is started, nothing will stop the ardor of these children. Whether it is bees, birds, butterflies or bugs, get a class of children enthused, and you will have results that last a life time even though the noise of enthusiasm for the subject wanes. So by applying this psychology, we have worked out what we call "The Iowa Plan for Dental Service," with excellent results, as you will agree when the facts and figures show that, our first year, we had 56 per cent of sound teeth and the fourth year shows a gain of 78 per cent. The children are interested. As many as could had all the cavities filled and other necessary work done. The question to be answered is: "How can we keep them interested?" for they must have continued stimulus until tooth care becomes a habit.

In Waterloo, "The Iowa Plan for Dental Service" and the organization of the "Tooth Brush Club" answered this question.

The Iowa Plan for Dental Service has been carefully worked out by the Oral Hygiene Committee of the Iowa State Dental Society. It has been used successfully in city, town and country schools all over Iowa. According to this plan, the dentists do not enter the school, but they do the part of the work that no one but a skilled dentist can do. They remain in their offices and fill the teeth of hundreds of pupils sent to them every year by the dental hygienists, principals and teachers. There are five distinct parts to the program:

1. Examination of the teeth of each child by the dental hygienist with the mouth mirror and explorer. This examination upsets the school curriculum very little and is made with little loss of time. Any evidences of decay, stain, irregularity of the teeth or inflammation or swelling of the gums is sufficient cause for sending the child to his dentist for closer examination and attention. Such instructions for further attention are contained on the "blue notification card" that is given the child, which he is instructed to take home to his parents and then to his dentist. When the necessary work is completed, the dentist signs the card and it is returned to the teacher by the child. This card, properly signed and returned to the teacher, shows that the child has had the necessary dental attention, and makes it possible for the teacher to keep a complete dental record of all her pupils. The teacher keeps the record on a chart especially prepared for that purpose, called the "Room Record Chart." It is used by the hygienist at the time the examination is made to record the condition of each child's mouth and thereafter to credit the child with having the needed work done when he returns his card signed by the dentist. This chart gives, at a glance, a complete picture of the condition of the mouth of the child. Another chart, called the "Dental Honor Roll," is used to record the names of those who have brought their cards from the dentist properly signed It also serves as a means of checking the progress made in getting the pupils to obtain necesary dental attention. The effect on the pupils is twofold. Those who have not their names on the honor roll will do all that they can to get it there; those who have their names on the honor roll will do everything they can to induce the lagging ones to get their names on also.

In other words, the "Dental Honor Roll" will help to develop a room pride in 100 per cent healthy mouths.

2. Classroom talks by the dental hygienist on food and cleanliness, the importance of getting cards signed by the dentist and returned to the teacher as soon as possible, the value of good teeth, and the importance of brushing the teeth three times a day and visiting the dentist regularly so that their teeth are always in good repair.

3. Individual instruction, such as check-ups, follow-ups and toothbrush drills.

4. Group instruction: mothers' meetings, parent-teacher association meetings and presentations of health films, etc.

5. Attention to the pre-school child, which offers a wonderful opportunity to further bolster up the program of prevention. (In Waterloo, the past year, we began the work of prevention with the pre-school children; and we have in our schools today five 100 per cent kindergartens, with the others ranking close in the race for good teeth.)

TOOTHBRUSH CLUB

No child was eligible for membership unless he owned a good toothbrush, such as was advised, used it three times a day, and had had all reparative work taken care of which was checked against him during the first examination. As a symbol of this child's rating in mouth health, he was given a pin which he was proud to show his fellow-students. Unhappy indeed was the child who could not earn the right to wear this pin. One little fellow had a toothbrush which he used regularly. He also had a cavity in a first molar, so could not become a member of the toothbrush club, even though his heart ached over his outcast state. He had one lonely dollar, which he had saved penny by penny, and this small fortune (for such it was to him) he took to a dentist and asked if he would fix that tooth for a dollar. He met with refusal. But not believing the verdict to be final, he went to another, with the same result. Still, undaunted, he walked into the office of an old practitioner, who, with a twinkle in his eye and a world of sympathy in his heart, asked the youngster to come in, and fixed his tooth for a dollar. Time couldn't fly fast enough until the dental hygienist checked over this school again. And how proudly he displayed his pin to his fellow-pupils!

This is only one of the myriad living instances of the earnestness of the boy and girl crusaders which have come to my attention during the school year. To be sure that the practice of oral cleanliness has become a habit, a child could be dropped from the club if he did not keep his mouth up to the standard mark.

TOOTHBRUSH DRILLS

The principal of the school to be visited was called by phone and told to announce that each child was to bring his toothbrush, carefully wrapped in a piece of paper and sealed in an envelope. The toothbrush drill was given to each child. Five or six children were called from a room at one time and taken out in the hall, or into the kitchen, where water was available. Their toothbrushes were examined. If the brush was not the right kind, or a new one was needed, it could be purchased from the hygienist at cost. Then they were not only told how to brush their teeth, but went through the process under supervision. All mouths needing dental attention were checked over to see if the work had been done. If the parents had not responded, or if the response was un-

favorable, a home call followed. Where financial conditions made it impossible for parents to have this work done, the children were taken to dentist in the city who had generously volunteered to do such work as this to help the children to membership in the toothbrush club. In most of the cases, their efforts were heartily appreciated.

Nothing is worth doing unless one meets enough difficulties to cause some thinking on the subject. So it is with oral hygiene in Waterloo. We have the cooperation of the superintendent, principals, teachers and pupils; but the cooperation of the mothers and fathers was not so easy to gain. The most common answer to a request to have a child's teeth fixed was: "I will have the teeth fixed as soon as possible"; which might mean next week or next year. If "as soon as possible" had not arrived by the time the school was gone over again, the dental hygienist called in person to emphasize the need of immediate action.

Again, there is the class of vain parents who will not admit that their offspring has any physical defects outside their knowledge. They seem to feel that they know all that is necessary to guide their child in the paths of good health. Of course, this condition resulting from a lack of proper knowledge had to be adjusted by a personal call. A large percentage of the parents who were told that the baby teeth needed filling responded that they would soon be lost, so that work would not be necessary. This belief is hard to overcome. You are familiar with the errors made in mistaking the first permanent molars for a deciduous tooth. We hope to gradually dispel this impression.

With the splendid help and cooperation that we have received from the dentists of Waterloo, and the constant watchfulness for indications of carelessness and persistent checking up on the part of the dental hygienist, we have been able to accomplish the following:

1924-1925: Examination of 2,426 children for dental defects during September and October showed that 32 per cent had teeth in good condition. The final recheck in June revealed that 56 per cent had teeth in good condition.

1925-1926: Examination of 2,555 children for dental defects during September and October showed that 51 per cent had sound teeth. The final recheck in June revealed that 81 per cent had sound teeth.

1926-1927: Examination of 2,604 children for dental defects during September and October showed 48 per cent to have sound teeth. In June, the final recheck showed that 80 per cent had sound teeth.

1927-1928: Examination of 2,771 children during September and October for dental defects showed that 49 per cent had sound teeth. In March, a recheck showed that 78 per cent had sound teeth.

Having considered the results that we have been able to get and the main problems that we have been called on to solve, let us look into the future of preventive dentistry. Not until the gospel of preventive dentistry is taught and the habit of oral cleanliness becomes a constant habit to the boys and girls who are now in our public schools will the interest of the parents be aroused in preventive dentistry. We must reach the parents through the children. If the boys and girls now in our care can learn the joy and happiness that comes from good health and clean living, we need not worry about the future generations. The

responsibility of preventive dentistry rests heavily upon the dentists and dental hygienists because they have the knowledge which can save mouths from destruction. We must teach these boys and girls that, in breaking the laws of nature, they are injuring themselves. Let us not think of them as a group. Let us think of them as individuals, each one different from the other.

DIRECTORY OF STATE SOCIETY OFFICERS AND DATES OF MEETINGS

ALABAMA

J. G. Hopping, President.....Bankers Bond Bldg., Birmingham
F. F. Perry, Secretary-Treasurer.....Montgomery
Date of next meeting: April 16-18, 1929.
Place of next meeting: Birmingham.

ARIZONA

R. Dean Creamer, President.....Goodrich Bldg., Phoenix
Robert A. McCall, Secretary-Treasurer.....Goodrich Bldg., Phoenix
Date of next meeting:
Place of next meeting:

ARKANSAS

Nolan Helstern, President.....Springdale
I. M. Sternberg, Secretary-Treasurer.....Fort Smith
Date of next meeting:
Place of next meeting: Little Rock.

CALIFORNIA (STATE)

B. Frank Gray, President.....209 Post St., San Francisco
F. T. West, Secretary.....2180 Washington St., San Francisco
Louis Graham, Treasurer.....516 Sutter St., San Francisco
Date of next meeting:
Place of next meeting: (Will meet with Pacific Coast Dental Conference, San Francisco, Calif., July 8-12, 1929.)

CALIFORNIA (SOUTHERN)

Charles C. Noble, President.....1741 Crenshaw Blvd., Los Angeles
Charles M. Alderson, Secretary.....Edwards & Wildey Bldg., Los Angeles
F. G. Staley, Treasurer.....Roosevelt Bldg., Los Angeles
Date of next meeting:
Place of next meeting:

COLORADO

Earl H. Zimmer, President.....Exchange Nat. Bk. Bldg., Colorado Springs
Lynn D. Mathews, Secretary.....Republic Bldg., Denver
Oliver H. Devitt, Treasurer.....Majestic Bldg., Denver
Date of next meeting: June 17-19, 1929.
Place of next meeting: Colorado Springs.

CONNECTICUT

Joseph W. Borchardt, President.....108 Bank St., Waterbury
Clarence E. Peterson, Secretary-Treasurer.....34 Park Place, Rockville
Date of next meeting: April 30-May 2, 1929.
Place of next meeting: Waterbury.

DELAWARE

Clyde A. Nelson, President.....Milford
William Stewart, Jr., Secretary.....Medical Arts Bldg., Wilmington
P. A. Traynor, Treasurer.....807 Washington St., Wilmington
Date of next meeting:
Place of next meeting:

DISTRICT OF COLUMBIA

C. Willard Camalier, President.....1029 Vermont Ave., Washington
W. M. Simkins, Secretary.....720 Woodward Bldg., Washington
M. F. Finley, Treasurer.....1928 I St., N. W., Washington
Date of next meeting:
Place of next meeting:

FLORIDA

F. L. Adams, President.....608 Tampa St., Tampa
C. C. Stewart, Secretary-Treasurer.....Hall Bldg., St. Petersburg
Date of next meeting:
Place of next meeting:

GEORGIA

H. H. Williamson, President.....Albany
G. A. Mitchell, Secretary.....Candler Bldg., Atlanta
B. K. Vann, Treasurer.....Candler Bldg., Atlanta
Date of next meeting: June 12-14, 1929.
Place of next meeting: Atlanta.

HAWAIIAN ISLANDS

John Knight, President.....Damon Bldg., Honolulu
H. P. Choy, Secretary.....4 King St., Honolulu
Joseph Pekelo, Treasurer.....471 S. Berentania St., Honolulu
Date of next meeting:
Place of next meeting:

IDAHO

R. G. Ioset, President.....Twin Falls
H. B. Colver, Secretary.....Eastman Bldg., Boise
T. L. Cartney, Treasurer.....Filer
Date of next meeting:
Date of next meeting:

ILLINOIS

Albert E. Converse, President.....Ridgely Farmers Bk. Bldg., Springfield
A. B. Patterson, Secretary.....Morris Bldg., Joliet
E. L. Burroughs, Treasurer.....Edwardsville
Date of next meeting: May 14-16, 1929.
Place of next meeting: Springfield.

INDIANA

W. R. Meeker, President.....Wallace Theater Bldg., Peru
C. L. Byers, Secretary.....2138 N. Illinois St., Indianapolis
W. C. Hessler, Treasurer.....Ben Hur Bldg., Crawfordsville
Date of next meeting: May 20-22, 1929.
Place of next meeting: Indianapolis.

IOWA

Earle S. Smith, President.....613 E. Court St., Iowa City
John Scholten, Secretary.....American Trust Bldg., Cedar Rapids
Frank Fourt, Treasurer.....Fairfield
Date of next meeting: May 7-9, 1929.
Place of next meeting: Cedar Rapids.

KANSAS

Homer B. Robison, President.....Hutchinson
Fred A. Richmond, Secretary..Federal Reserve Life Insurance Bldg., Kansas City
W. T. Triplett, Treasurer.....Salina
Date of next meeting: April 29-May 2, 1929.
Place of next meeting: Wichita.

KENTUCKY

George H. Means, President.....1380 Bardstown Road, Louisville
Frank B. Hower, Secretary.....323 E. Chestnut St., Louisville
Fred Millard, Treasurer.....Louisa
Date of next meeting: April 9-11, 1929.
Place of next meeting: Louisville.

LOUISIANA

L. J. Schoeny, President.....Donaldsonville
B. J. LaCour, Secretary.....Welsh
O. J. Ory, Treasurer.....Garyville
Date of next meeting:
Place of next meeting: Alexandria.

MAINE

William D. Taylor, President.....Mechanic Falls
Wallace F. Fogg, Secretary.....Yarmouth
Irving E. Pendleton, Treasurer.....Lewiston
Date of next meeting: June 20-22, 1929.
Place of next meeting: Portland.

MARYLAND

E. W. Swinehart, President.....Medical Arts Bldg., Baltimore
Norval H. McDonald, Secretary.....Medical Arts Bldg., Baltimore
Walter E. Green, Treasurer.....2958 W. North Ave., Baltimore
Date of next meeting: May 6-8, 1929.
Place of next meeting: Baltimore.

MASSACHUSETTS

Charles W. Hammett, President.....2½ Main St., Taunton
William H. Gilpatric, Secretary.....358 Commonwealth Ave., Boston
Joseph T. Paul, Treasurer.....419 Boylston St., Boston
Date of next meeting: May 6-10, 1929.
Place of next meeting: Boston.

MICHIGAN

P. C. Lowery, President.....8545 Gratiot Ave., Detroit
W. R. Davis, Secretary.....State Department of Health, Lansing
E. J. Chamberlain, Treasurer.....Ashton Bldg., Grand Rapids
Date of next meeting:
Place of next meeting:

MINNESOTA

R. G. Keyworth, President.....Lowry Bldg., St. Paul
George D. Estes, Secretary.....Yeates Bldg., Minneapolis
T. D. Abernathy, Treasurer.....Rochester
Date of next meeting: March 6-8, 1929.
Place of next meeting: St. Paul.

MISSISSIPPI

C. K. Bailey, President.....Grenada
C. A. Moore, Secretary.....Lexington
J. H. Brown, Treasurer.....Newton
Date of next meeting:
Place of next meeting: Jackson.

MISSOURI

C. B. Coleman, President.....Poplar Bluff
C. W. Digges, Secretary.....Columbia
J. F. McLellan, Treasurer.....Moberly
Date of next meeting: May 12-14, 1929.
Place of next meeting: St. Joseph.

MONTANA

T. P. Regan, President.....Helena
J. E. Buehler, Secretary-Treasurer.....Great Falls
Date of next meeting: March 28-30, 1929.
Place of next meeting: Butte.

NEBRASKA

H. A. Nelson, President.....Omaha Loan Assn. Bldg., Omaha
H. E. King, Secretary.....Peters Trust Bldg., Omaha
G. L. Spencer, Treasurer.....Sharp Bldg., Lincoln
Date of next meeting: May 20-23, 1929.
Place of next meeting: Omaha.

NEVADA

George A. Carr, President.....Farmers & Merchants Bk. Bldg., Reno
P. H. Phillips, Secretary-Treasurer.....Medico-Dental Bldg., Reno
Date of next meeting: June 1, 1929.
Place of next meeting: Reno.

NEW HAMPSHIRE

A. W. Woodman, President.....Plymouth
W. J. Moyles, Secretary.....1061 Elm St., Manchester
W. A. Young, Treasurer.....40 N. Main St., Concord
Date of next meeting:
Place of next meeting:

NEW JERSEY

A. E. Boice, President.....Broad St. Bk. Bldg., Trenton
F. K. Heazelton, Secretary.....223 E. Hanover St., Trenton
George H. Griffith, Treasurer.....42 W. State St., Trenton
Date of next meeting:
Place of next meeting:

NEW MEXICO

E. G. Brentari, President.....Belen
Fred Muller, Secretary-Treasurer.....Taos
Date of next meeting:
Place of next meeting: Las Cruces.

NEW YORK

Gerald G. Burns, President.....Professional Bldg., Rochester
A. P. Burkhart, Secretary.....57 E. Genesee St., Auburn
George H. Butler, Treasurer.....Skaneateles
Date of next meeting: May 15-17, 1929.
Place of next meeting: Rochester.

NORTH CAROLINA

I. R. Self, President.....Lincolnton
Dennis Keel, Secretary-Treasurer.....Greensboro
Date of next meeting:
Place of next meeting: Wrightsville Beach.

NORTH DAKOTA

R. W. Pote, President.....Fargo
L. I. Gilbert, Secretary.....Fargo
L. E. Musberger, Treasurer.....Jamestown
Date of next meeting:
Place of next meeting: Grand Forks.

OHIO

J. Walter Hartshorn, President.....Nicholas Bldg., Toledo
E. C. Mills, Secretary.....255 E. Broad St., Columbus
Harry Cope, Treasurer.....327 E. State St., Columbus
Date of next meeting: December 4-6, 1928.
Place of next meeting: Toledo.

OKLAHOMA

W. J. Scruton, President.....First Nat. Bk. Bldg., Oklahoma City
E. E. Sanger, Secretary.....Yukon
C. A. D. Beer, Treasurer.....Enid
Date of next meeting:
Place of next meeting: Oklahoma City.

OREGON

A. J. Brock, President.....Selling Bldg., Portland
F. W. Hollister, Secretary.....Morgan Bldg., Portland
M. Arch Milligan, Treasurer.....Stevens Bldg., Portland
Date of next meeting:
Place of next meeting: (Will meet with Pacific Coast Dental Conference, San Francisco, Calif., July 8-12, 1929.)

PENNSYLVANIA

W. H. Fordham, President.....Connell Bldg., Scranton
C. W. Hagan, Secretary.....1011 Macon Ave., Pittsburgh
W. L. Fickes, Treasurer.....East End Savings & Trust Bldg., Pittsburgh
Date of next meeting:
Place of next meeting:

PORTO RICO

Jose Vincente, President.....San Justo 33, San Juan
Jose Hernandez Gonzalez, Secretary.....Cruz St. 15, San Juan
Ramon Fernandez Carballo, Treasurer.....Stop 19, Santurce
Date of next meeting:
Place of next meeting:

RHODE ISLAND

B. Cecil Burgess, President.....56 Snow St., Providence
H. F. McKanna, Secretary.....15 Washington St., West Warwick
Thomas S. MacKnight, Treasurer.....130 Touro St., Newport
Date of next meeting:
Place of next meeting: Providence.

SOUTH CAROLINA

C. C. Fuller, Jr., President.....Greenwood
T. C. Sparks, Secretary.....Greer
G. M. Mears, Treasurer.....1507 Main St., Columbia
Date of next meeting:
Place of next meeting: Columbia.

SOUTH DAKOTA

Robert Jasman, President.....Sioux Falls
Ernest W. Elmen, Secretary.....Sioux Falls
J. W. King, Treasurer.....Aberdeen
Date of next meeting: July 1-2, 1929.
Place of next meeting: Rapid City.

TENNESSEE

William F. Stone, President.....Volunteer State Life Bldg., Chattanooga
T. J. Reavis, Secretary-Treasurer.....Lawrenceburg
Date of next meeting: June 13-15, 1929.
Place of next meeting: Chattanooga.

TEXAS

T. F. Cox, President.....Scarborough Bldg., Austin
J. G. Fife, Secretary-Treasurer.....Medical Arts Bldg., Dallas
Date of next meeting: April 16-19, 1929.
Place of next meeting: Beaumont.

UTAH

R. C. Dalgleish, President.....Deseret Bank Bldg., Salt Lake City
T. Fred Hardy, Secretary-Treasurer.....Medical Arts Bldg., Salt Lake City
Date of next meeting:
Place of next meeting: (Will meet with Pacific Coast Dental Conference, San Francisco, Calif., July 8-12, 1929.)

VERMONT

George D. Samson, President.....Burlington
Lloyd C. Robinson, Secretary.....Morrisville
H. F. Killary, Treasurer.....Burlington
Date of next meeting: May 22-24, 1929.
Place of next meeting: Burlington.

VIRGINIA

R. C. Walden, President.....321 West Franklin St., Richmond
A. M. Wash, Secretary-Treasurer.....Medical Arts Bldg., Richmond
Date of next meeting:
Place of next meeting:

WASHINGTON

J. W. Trosper, President.....Cobb Bldg., Seattle
Will G. Crosby, Secretary-Treasurer.....Medical-Dental Bldg., Seattle
Date of next meeting:
Place of next meeting: (Will meet with Pacific Coast Dental Conference, San Francisco, Calif., July 8-12, 1929.)

WEST VIRGINIA

Frank L. Wright, President.....Wheeling
J. E. Herbert, Secretary.....Martinsburg
John S. Stone, Treasurer.....Clarksburg
Date of next meeting: May 27-29, 1929.
Place of next meeting: White Sulphur Springs.

WISCONSIN

J. W. Crawford, President.....First Wis. Nat. Bk. Bldg., Milwaukee
R. W. Huegel, Secretary.....104 King St., Madison
H. S. Huxtable, Treasurer.....Mineral Point
Date of next meeting: July 9-11, 1929.
Place of next meeting: Milwaukee.

WYOMING

G. A. DeFreece, President.....Midwest
E. C. Andrew, Secretary.....Citizens Bank Bldg., Cheyenne
Peter Appel, Jr., Treasurer.....Hynds Bldg., Cheyenne
Date of next meeting:
Place of next meeting: Cheyenne.

CURRENT LITERATURE

EXTRACTS AND ABSTRACTS

Mercurochrome and Iodin as Disinfectants of the Mucous Membrane of the Mouth: Experiments undertaken with a view to ascertaining the relative efficacy of mercurochrome and iodin solutions as disinfectants of the oral mucous membranes are reported by F. E. Rodriguez (*J. A. M. A.*, Sept. 8, 1928). The following antiseptics were employed in the tests: 1. Mercurochrome-22 soluble (dibromoxymercuriflourescein), a commercial preparation sold generally and labeled "For use as a general antiseptic in place of iodin." This antiseptic is a 2 per cent aqueous solution of mercurochrome. 2. Mercurochrome, 5 per cent, in 50 per cent alcohol. 3. Mercurochrome-alcohol-acetone solution. This is prepared by dissolving 2 gm. of mercurochrome in 35 c. c. of water and adding 55 c. c. of 95 per cent alcohol and 10 c. c. of acetone. 4. Alcohol-acetone solution, which consists of alcohol, 95 per cent, 55 c. c.; distilled water, 25 c. c.; acetone, 10 c. c. 5. Iodin-glycerin, 3.5 per cent, made up of equal parts of tincture of iodin, U. S. P., and chemically pure glycerin. 6. Iodin-glycerin, 1.75 per cent, consisting of iodin solution in the proportion of one-fourth of the tincture and three-fourths of the chemically pure glycerin. 7. Iodin-glycerin, 0.875 per cent, made up of iodin solution in the proportion of one-eighth of the tincture and seven-eighths of chemically pure glycerin. 8. Iodin, 3.5 per cent in alcohol. Each solution was tested directly on the surface of the moist oral mucous membrane of ten different persons, with the exception of number 8, which was tested six times, and number 4, which was tested only four times. On the basis of the results of these experiments, the following conclusions were reached: 1. Mercurochrome-220 soluble (2 per cent aqueous solution) is too feeble an antiseptic to be used safely as a surface disinfectant of the oral mucous membranes. 2. The 5 per cent mercurochrome solution in alcohol and the mercurochrome-alcohol-acetone preparations both possess decided advantages over the aqueous solution, but fail in too large a proportion of cases to be considered effective in surface disinfection of the oral mucous membrane. 3. Iodin in dilutions of 3.5 per cent, and even in 1.75 per cent strength, preferably in glycerin, is an effective germicide from the standpoint of surface disinfection of the oral mucous membranes.

The Heart During Anesthesia: A discussion of the heart during anesthesia and operative procedures is presented by H. M. Marvin (*New England J. Med.*, Sept. 20, 1928), in an endeavor to clear up the confusion of the young surgeon as to what criteria should be taken as a guide in surgical cases complicated by heart conditions. Anesthesia by means of the gases, ethylene and nitrous oxid, alone is considered, chloroform being omitted as being little used in this country. The following conclusions are reached: There is no convincing evidence that anesthesia and operation, in themselves, have any damaging action on the normal heart. A damaged heart, whatever its physical signs, is the equivalent of a normal one for anesthesia and operation if it is carrying on an adequate circulation under normal conditions of life; with the possible exception of the syphilitic heart. The history, therefore, is more important than the physical examination, although both should be had. Patients with heart failure and those with auricular fibrillation, even without heart failure, should receive digitalis before operation if possible. The only important arrhythmias are auricular fibrillation and heart block. Digitalis should be given in therapeutic doses or not at all. It should not be given as a routine preoperative measure. Of the available general anesthetics, etheylene is probably the one that has most to commend it for use in patients with heart failure. Finally, and possibly most important of all,

it is far more important to select the proper anesthetist than the proper anesthetic.

Effects of Ethylene-Oxygen Anesthesia on the Normal Human Being: Fifteen men, eight of whom were graduate physicians, officers of the medical corps, and seven of whom were enlisted men of the medical department, volunteered to be anesthetized for this study, which is reported by John D. Brumbaugh (*J. A. M. A.*, Aug. 18, 1928). The age range was 21-37, and all participants were pronounced normal after a physical examination. All stated that the induction sensation was pleasant, and without a sense of suffocation. None mentioned the odor of the gas during induction. Average time of recovery was one minute and forty-four seconds. There was no change in the hemoglobin, as tested immediately and again twenty-four hours after anesthesia. No appreciable destruction of red blood cells and no appreciable derangement of the biliary system occurred. Marked increase in blood sugar which occurred immediately after anesthesia was followed by a return to normal in about twenty-four hours. Blood urea showed an increase after twenty-four hours. There was a moderate decrease in the carbon dioxid combining power of the blood, with return to normal in twenty-four hours. There was no change in the coagulation time of the blood and no change in the character of the blood clot. No urinary changes were noted. The systolic blood pressure increased, as did the diastolic, the latter slightly. The pulse pressure increased appreciably, but there was no appreciable increase in the rate. Nine of the subjects complained of headache following anesthesia and thirteen were nauseated, twelve markedly. There were no respiratory complications and no gas pains. The increased morbidity may be accounted for by the absence of postanesthetic control and longer duration of the anesthesia than is usual in operative cases.

Factors in the Decay of the Teeth: After an annual inspection of the school children of Rochester, Minn., in which a high incidence of dental caries was revealed, Louise O. Kappes (*Am. J. Dis. Child.*, August, 1928), in an effort to evaluate the possible etiologic factors, selected for comparison twenty-five children with perfect or nearly perfect teeth and twenty-five with marked dental caries. The ages ranged from 5 to 13. The average age in the first group was 9.5, in the second, 6.1. The question thus raised as to whether the first teeth decay more readily than the second cannot be answered from this study. Variation in health of the mothers in the prenatal period was too small to be of any significance. Six mothers in each group had marked dental trouble during pregnancy. Green vegetables were predominant in the prenatal diet of fourteen mothers of the first group and ten of the second. Meat, potatoes and sweets predominated in the diet of six of the first group and ten of the second. Four of the children of Group 2 were prematurely born. Eleven in each group were breast fed for more than nine months, and four, for less than one month. Cow's milk was used almost entirely for complementary or supplementary feedings. Milk preparations were used a few weeks in several cases, but in only one (poor teeth) for the whole nursing period. Cereal feedings were started before the age of 9 months in eleven of the first group and in sixteen of the second. Vegetables were regularly given before the age of 1 year to fourteen of each group and before the age of 9 months to four of the first group and two of the second. The number receiving orange juice was too small to be significant. The same is true of cod liver oil. Thirteen of the first group and eight of the second cut their first teeth before 7 months. After 1 year of age, green vegetables, fruit, meat and eggs predominated in the diet of thirteen of the children with good teeth and in only three of those with poor teeth; whereas, carbohydrates predominated in the diet of fourteen with poor teeth and of only three of those with good teeth. Fourteen of those with poor teeth and only two of those with good teeth ate a great deal of candy and other sweets. The findings regarding consumption of milk, carbohydrates and vegetables in the two groups suggest that milk, when served in fairly large quantity, is more likely to crowd out vegetables and fruit than carbohydrates, and yet that the milk is not so effective as the fruits and vegetables in preventing dental decay. The mothers of ten children, four of the first group and six of the second, cleaned the teeth daily during the first three or four years. Regular brushing of the teeth one or more times daily was carried out by nine of the group with good teeth and fifteen of those with poor teeth. Outdoor play was approximately the same in each group. The general

health of twenty-two of the children with good teeth and of nineteen with poor teeth was considered good. The total number of diseases experienced was eighty-two in the first group and 104 in the second, although the children in the latter group averaged several years younger than in the first. Individual data indicate that infectious disease is significant in decay of the teeth. Heredity seems to be of no significance. In fact, the difference in the data obtained in the two groups is not so striking as one would expect. The only feature that seems of definite etiologic significance in preventing decay of teeth is a diet comosed largely of fruits and vegetables.

Clinical Experience with Irradiated Ergosterol: Irradiated ergosterol is found by Hess and Lewis (*J. A. M. A.*, Sept. 15, 1928) to be by far the most potent of the antirachitic agents. This sterol was differentiated from cholesterol, which was formerly supposed to be the substance which undergoes change in the irradiation of such foods as oils, milk and cereals by Tanret, a French chemist, in 1890, who showed that it could be extracted from fungi, yeast and mushrooms, particularly from ergot, a fungus that develops on grain. Hess and Lewis pronounce it an absolute specific, as compared to cod-liver oil, which, in the amount in which it can be given, is a specific of limited dependability, only moderately effective for the average infant, uncertain in action for the rapidly growing infant and ineffective for the premature. Irradiated ergosterol is found to be quite as valuable in tetany as in rickets, and in both disorders is remarkable for the rapidity as well as for the reliability of its action. The authors state that because of insufficient clinical experience, it is impossible to define its proper dosage as yet. Also, the various preparations cannot be evaluated, as they have not yet been assayed on the basis of the number of curative units (rat) which they contain. The amounts now recommended and employed are unnecessarily high, as shown by the fact that they induce an excess of calcium and inorganic phosphorus in the blood in the normal as well as in the rachitic infant—hypermineralization. Too great emphasis has been laid on rapidity of action. It would seem advisable, before distributing unreservedly to the medical profession this potent concentrate, to make a more thorough clinical study of its dosage, as has been done in regard to other potent extracts. In view of its reliability, its high degree of activity and its ease of administration, irradiated ergosterol should prove a most valuable addition to the rapidly increasing fund of specific antirachitic agents.

Agranulocytic Angina: A case of agranulocytic angina in a woman, aged 43, is reported by Tyree C. Wyatt (*New England J. Med.*, Sept. 13, 1928). After a period of general malaise, during which several furnucles developed on the face and hands, the patient was admitted to the hospital complaining of sore throat. The following day, a tender lump became evident under the left jaw. Headache and rise in temperature were attendant symptoms. Later, localized areas of infection were evidenced in the left infraclavicular and left axillary regions. Surgical drainage resulted in subsidence of all symptoms. The bacteriologic findings in the case were inconclusive. The blood culture was negative. There was at all times a low leukocyte count. Cultures from the throat showed no evidence of diphtheria bacilli, Vincent's organisms or fungi. Both throat smears and pus drained from the abscesses showed streptococci and staphylococci, as well as gram-positive bacilli.

Vincent's Infection: The true diagnosis of Vincent's infection can be made only in a medical or dental laboratory with the use of the microscope. Whenever the uncomfortable symptoms of a persistent sore mouth are experienced, that person should immediately consult his dentist or his physician for treatment. Vincent's infection cannot be treated at home with household remedies. In the first place, it could not be properly diagnosed, and the danger of its becoming acute, with serious after-results, would be great. In case a dentist, or a physician is not available, the treatment should be the use of a mouth wash at frequent intervals until skilled treatment can be obtained. Undoubtedly, the micro-organism causing this disease cannot live or keep its virulent nature in the presence of air or free oxygen. Although it might continue to exist for sometime in the presence of oxygen, it will not multiply. It is evident, then, that any oxygen-liberating compound will check the growth of these organisms. For this purpose there are three outstanding drugs which may be used as an efficient mouth wash. They are

hydrogen peroxide, potassium permanganate and sodium perborate. All of these drugs, when freshly prepared, liberate oxygen. Hydrogen peroxide is a solution easily obtained and is capable of liberating ten volumes of oxygen. When used as a mouth wash, diluted with equal volumes of water and used at frequent intervals, forcibly working it back and forth in the mouth by contraction of the lips and cheek muscles, it will serve very well as a temporary measure. Potassium permanganate in 1:5,000 dilution is good, although it has a disagreeable taste. Sodium perborate is a white powder readily soluble in water, and when freshly dissolved liberates about 9 per cent oxygen. It is a good home remedy, but will not keep in solution and will lose strength in solid form if kept in a warm place. Any of these remedies will serve as a temporary expedient for a few days until the services of a dentist can be secured; but it is dangerous to delay treatment if it can be possibly obtained. During the acute stage, the toothbrush should not be used, as it is likely to cause a hemorrhage by forcibly removing the diseased membrane. This would furnish more pabulum for bacterial growth. Although the outlook in Vincent's infection is favorable and the condition usually responds quickly to competent treatment, neglect may result seriously, as the micro-organisms causing this disease have been isolated from other more serious diseases such as meningitis, peritonitis and diabetes. Most cases of Vincent's infection can be cured with proper treatment. — *United States Public Health Service.*

Foreign Literature

Concerning Teeth in Dermoid Cysts of the Ovaries (Dr. Kanamori, Vierteljahrschr. f. Zahnheilk., 44, No. 2, 1928): This is an exhaustive microscopic, roentgenographic and histologic study of fifty ovarian dermoid cysts made at the Dental Institute of Breslau, Germany. Of these fifty cysts, fourteen were found to contain teeth, for the most part anatomically well-formed, but about one-third smaller than the normal in size. All the teeth were represented: incisors, cuspids, bicuspids and molars, with the last predominating. Two was the smallest number of teeth found in any one cyst; but more often the number was four, and one cyst contained six teeth, four of which were large well-formed molars. Six roentgenograms and eight excellent photographs illustrate the article, the latter showing the teeth, hair and other contents of the cysts in natural size, and in splendid detail. A continuation of this article will follow in subsequent numbers.

James D. Frankel, Chicago.

Focal Infections and Their Relation to Mental Diseases (William Hunter, Vierteljahrschr. f. Zahnheilk., 44, No. 2, 1928):* That focal infections should be more widely recognized as an etiologic factor in mental disorders is the lesson that Dr. Hunter brings in this article. The number of "septic psychoses" in the insane asylums of the world is greater, he says, than that of any other single group of hospital patients. It is a most interesting and dramatic recital of extreme individual cases and statistical records from his own experience, and from that of Dr. Cotton of the New Jersey State Hospital for the Insane, and of Dr. Graves of Birmingham, England. Dr. Hunter makes a convincing plea for the early diagnosis and removal of all forms of focal infections, not only as a curative but also as a prophylactic measure. Of particular interest to us, as dentists, is the fact that, in a list of the most important focal infections, he places oral sepsis first, infected tonsils second, infections of nasal and mastoid cells third, those of the cervix uteri fourth, and of the colon fifth. Of the oral foci, he stresses as particularly "virulent" the chronic conditions: impacted, unerupted and pulpless teeth, root fragments and pyorrhea.

James D. Frankel, Chicago.

Dunlop's Method for Treatment of Diffuse Atrophy and Gingivitis Ulcerosa (B. Gottlieb, Ztschr. f. Stomatol., No. 1, 1928, p. 20): In Dunlop's method, a paraffin is used chiefly mixed with boracic acid, a paste consisting chiefly of glycerin and boracic acid and oxygen mixed with medicaments introduced parenterally into the tissue. The method has been proved satisfactory in the treatment of ulcerative gingivitis (stomacace) and of acute diffuse atrophy of the aveolar bone. Especially, the gingivitis hypertrophicans connected with the last mentioned disease, characterized by the lowering of the specific dynamic efficacy of albumin and by the prevalence of plasma cells in the histologic pic-

*This article also appeared in The American Dental Surgeon, vol. 48.

ture, disappears without any surgical procedure.

BALINT ORBAN, Chicago.

A Peculiar Disturbance of Speech in Postencephalitic Parkinson's Disease and Its Cure by Prosthetic Appliances (H. Herschmann and H. Wolf, Ztschr. f. Stomatol., No. 8, 1928, p. 813): Report is made of a case of postencephalitic Parkinsonism, which showed, as its chief symptom, a disturbance of speech caused by hyperkinesis of the tongue. The patient found help in biting heavily on a pipestem. Improvement in speech was brought about by means of adequately raising the bite. This was accomplished by a prosthetic appliance interposed between the molars and the lower jaw on each side.

BALINT ORBAN, Chicago.

Paradental Pyorrhea and Alveolar Atrophy (B. Gottlieb, Fortschr. d. Zahnheilk., No. 5, 398, 1928): Since the early description of the diagnostic symptoms of diffuse atrophy of the alveolar process, other noteworthy observations have been made. Within the past year, it has been observed (Weinmann) that a high percentage of patients affected with acute diffuse atrophy showed an approximately 20 per cent lowered specific dynamic effect on protein metabolism. In a study of experiments made on thirty dogs in which marked traumatic occlusion had been produced, many significant histologic changes were produced. These changes showed that epithelial proliferation along the cementum is not influenced by the Sharpey fibers but rather by the quality of the tooth surfaces. Neither is the development of cement hypoplasia explained upon the basis of the mechanical-functional theory of Haul and Lange. The manner in which cementum, dentin and bone are resorbed as well as repaired under various forms of excessive stresses strongly contradicts the advocates of the traumatic occlusion theory. The greater number of the cases of alveolar pyorrhea are of filth origin. They are treated by scaling and thorough mouth hygiene. The balance of the cases, in which tartar and filth are absent, offer the real difficulty. This type of pyorrhea is usually associated with diffuse atrophy. At the present time, marked success has been attained in the treatment of these cases by the Dunlop method.

EMIL MUELLER, Chicago.

Local Anesthesia (Guido Fischer, Fortschr. d. Zahnheilk., No. 1, 1928, p. 52): In anesthetic properties, tutocain closely approaches cocain. It is less poisonous than procain. On superficial application, it produces anesthesia closely approximating that produced by cocain. It threatens to displace cocain in this respect. It is well adapted to children's practice. A 10 per cent solution painted on the surface of the mucous membrane, three times at one minute intervals, will produce better anesthesia than will the injection of weak solutions. The anesthetic action of procain is greatly increased and the toxin effect materially reduced, if the procain solution is combined with a potassium sulphate solution. Anesthesia may be obtained in 41 per cent less time with this combination. Alkalinity or acidity have an important bearing on the anesthetic solution. The nearer the alkanlinity approaches the alkalinity of blood, the better will be the absorption and diffusion of the anesthetic solution. The poisonous effect will also be reduced and less reaction will follow the anesthesia. Every inflammation produces a local acidosis. The hydrogen ion concentration increases with the acuteness of the inflammation. Gaza and Brandi found that if a phosphate buffer solution with a p_H of 7.2-8.0 was injected, the effect was painless. If the p_H fell below 7.2, the effect was painful. The buffer solution consists of: 6.49 secund. sodium phosphate; 6.44 sodium chlorate; 1,000.00 distilled water. Porcelain prepared with this buffer solution and injected slowly at body temperature makes a much more satisfactory anesthetic solution. In combination, the work done formerly with a 4 per cent solution may be done with a 2 per cent solution. The 1 per cent solution will do the work of a 2 per cent procain solution. The epinephrin may also be reduced from 0.00005 gm. per cubic centimeter to 0.00003 gm. per cubic centimeter without changing the action.

EMIL MUELLER, Chicago.

The Fault of the Lower Jaw, Angle's Classifications II and III. A contribution to the classification of malocclusions. (A. Martin Schwarz, Ztschr. f. Zahnartl. Orthop.): The anatomic alterations of the jaws in Angle's classes II and III, noted by Sicher and Krasa and confirmed by Oppenheim, can be considered as adaptations to the exceptional functions of these malocclusions. As to the accom-

modation to a change of function, the lower jaw is the preinclined part of the entire jaw-mechanism. The shorter lower jaw in Class II is not too short, but just as long as required by function at the distal occlusion. The normal lower jaw would be too long in this case. The longer lower jaw is Class III is not too long for the altered function but of proper length; whereas, a normal lower jaw would prove to be too short. The short ramus mandibulae in Class II needs comparatively short chewing muscles with a large cross-cut, and leads therefore to a comparatively broad-faced (euryprosope) type. The long ramus in Class III needs comparatively long chewing muscles with a smaller cross-cut and produces, therefore, a comparatively long-faced (leptoprosope) type. The normal broad-faced type is only thus far connected with the pathologic type of Class II. The normal broad-faced type also has comparatively short and thick chewing muscles. The normal long-faced type has comparatively thin muscles. Through the distal displacement of the lower jaw, the broadness of the face is artificially increased, just as the calves of the legs are becoming fatter in consequence of wearing high heels. Also, in a normal jaw, a typical malocclusion can result only through the failure of the normal occlusion. This anomaly presents clinically the same aspect as a malocclusion produced by impediment of development in the jaw-bones already given in the germinal tissue. Referring to the foregoing, a genetic division of malocclusion into two parts is given: (*a*) primary, i. e., those caused through primary failure of the normal occlusion; (*b*) secondary, in which the altered occlusion is secondary. In the latter, an abnormal growth of the jaw bones is the primary cause. Mixed forms of both types, in which a trifling deviation from the normal growth of the participating bones has been the cause of failure of the normal occlusion, while further development of the anomaly, as a matter of course, is the result of orthodontic consequences of the malocclusion, are added to the forms of primary malocclusion. In general, we cannot blame the lower jaw for being the basis of primary malocclusion. On the contrary, the mobility of the lower jaw, as well as its accommodation to the altered mechanical situations, affords a fairly good function in cases of anomalous occlusion. Referring to the secondary malocclusions, the upper as well as the lower jaw can be at fault. Concerning the primary malocclusions, Angle's classification has proved to be on a genetic foundation.

BALINT ORBAN, Chicago.

Pathologic Physiology (Erich Grafe, Fortschr. d. Zahnheilk., No. 7, 1928, p. 651): The mechanism which regulates body temperature has been the basis of a great deal of speculation in the past. Through the work of Krehl and Isenschmid, it has been definitely established that the heat regulation of the body is controlled by a fixed center in the brain, that is, the tuber cinereum. In the event that this center is removed or destroyed, the warm blooded being becomes cold blooded. If a cannula is placed in this region and cold water is passed through the cannula, the body temperature rises. If warm water is passed through, the opposite takes place. From this center, the nervous impulses travel through the spinal cord to the organs where they bring about increased or decreased combustion. In consequence of nervous impulses from the brain, heat regulation takes place through physical and chemical means. Grafe and Freund have shown that impulses which influence heat regulation by the different means travel through different divisions of the cord. Chemical heat regulation takes place in the muscles and the liver, the physical means through the skin and sweat glands. Only when heat regulation is viewed from this new angle can the fever problem be properly comprehended. The manner in which foreign proteins in the blood produce fever is discussed. The question whether fever is a favorable or unfavorable reaction is also treated. There is much in support of the contentions of those who combat fever with antipyretics. The cold water treatment for typhus is defended with many statistics. Within the past two years, there have been added many worth while contributions on the nature of fever, but still the question as to its exact significance remains unanswered.

EMIL MUELLER, Chicago.

MISCELLANY

BOOK REVIEWS

Treatment of the Periodontal Pocket. By Harold Keith Box, D.D.S., Ph.D., Research Professor of Periodontology, Faculty of Dentistry, University of Toronto. 123 pages, illustrated with 120 cuts. Published by The University of Toronto Press.

It is well known that Dr. Box has given a great deal of attention to the study of this subject. For years, he has devoted his best energies to this one department of research, and this publication is the result. In 1924, he issued a report "Studies in Periodontal Pathology," which created considerable interest at the time, and the present volume is a continuation of that study.

In this later work, Dr. Box shows additional illustrations of restoration of alveolar bone after treatment of pockets, and the evidence would seem to prove the possibility of saving many teeth that in the past have been sacrificed. A careful study of this volume will well repay any practitioner who is interested in the subject.

Index of the Periodical Dental Literature, Published in the English Language, Including Ninety-Five Publications in England, Canada, Australia, New Zealand and the United States. For the years, 1921-1923. A *Classified Subject Index on the Plan of the Dewey Decimal Classification, an Alphabetical Author Index, a List of Dental Books Reviewed During the Same Period.* Compiled by Arthur D. Black, A.M., M.D., D.D.S., Sc.D., Dean, Northwestern University Dental School. Published by the Dental Index Bureau, 381 Linwood Ave., Buffalo, N. Y. Henry Kimpton, 263 High Holborn, London, England. 391 pages. Price, $6.00, except in foreign countries, where it is $6.50. This work is published under the auspices of the American Association of Dental Colleges, American Dental Association, Research Commission of the American Dental Association, British Dental Association, Canadian Dental Association, New Zealand Dental Association, and the Society of Dental Science of New South Wales.

Note is made of the fact that, on account of the vast amount of periodical literature, it has been found necessary to cut this volume from the usual five-year period to three years, a volume very little smaller than the previous five-year book resulting. The work involved in getting out these volumes cannot be comprehended by the average individual, but it is safe to say that no publication in dentistry will prove of greater permanent value than these.

A Practical Medical Dictionary of Words Used in Medicine with Their Derivation and Pronunciation, Including Dental, Veterinary, Chemical, Botanical, Electrical, Life Insurance and Other Special Terms; Anatomical Tables of the Titles in General Use, and Those Sanctioned by the Basle Anatomical Convention; Pharmaceutical Preparations, Official in the U. S. and British Pharmacopoeias and Contained in the National Formulary, and Comprehensive Lists of Synonyms. By Thomas Lathrop Stedman, A.M., M.D., Editor of the "Twentieth Century Practice of Medicine" and of the "Reference Handbook of the Medical Sciences," formerly editor of the "Medical Record." Tenth, Revised Edition. Illustrated, 1194 pages; price, $7.50. Published by William Wood and Company, New York.

Nearly 500 new medical terms have been included in this volume, and the book has been brought as nearly up to date as possible, by the elimination of many obsolete terms and the trade names of discontinued pharmaceutical preparations. Every progressive practitioner should have a reliable dictionary, and when the tenth edition of any work is called for it is *prima facie* evidence of its value.

Food, Nutrition and Health. By E. V. McCollum, Ph.D., Sc.D., and Nina Simmonds, Sc.D. (Hygiene), Professor, and Associate Professor, of Chemical Hygiene, School of Hygiene and Public Health, Johns Hopkins University, Baltimore, Md. Second

Edition, Revised. 148 pages; price, $1.00 postpaid. Published by the authors, East End Post Station, Box 25, Baltimore, Md.

When a book on this subject calls for reprinting three times and then for a second edition, it is evidence that it has created unusual interest. A book of this size treating the subject of diet in a nontechnical way will appeal to the average reader more than will a detailed treatment with all the scientific lore that has been gained, and this probably accounts for the fact that the book has enjoyed such a favorable reception on the part of the professions. We bespeak for it a continuance of the interest that has been taken in it since its first appearance.

LAST CALL!

The Commission for Scientific Research of the F. D. I. has decided, as the result of a motion of the president, Dr. Gottlieb, to announce a competitive research for a satisfactory root canal treatment, the winner of the contest to receive a gold medal and $1,000 in cash. The decision will be made at the International Congress of the F. D. I. in Paris in 1931.

The details of the contest cannot be described at this time. The attention of the profession is merely called to the contest and awards.

Proof of the value of the method used will be required. It is no longer sufficient that some one claims that his method is good, or the best; it must be proved.

After many years of experimenting, the Commission for Scientific Research of the F. D. I. offers a method of proof called by Dr. Gottlieb the "biologic method." The method has been worked out by B. Gottlieb, A. M. Schwarz and G. Stein.

This method is as follows:

Treat a tooth, fill the root canal and leave the tooth in the patient's mouth a few months. In some cases, it is desirable for clinical evidence to allow it to remain for six months. Then extract the tooth under aseptic conditions. Cut away the apex and, under aseptic conditions, implant it between the muscles of a rat. Let the rat live under good hygienic conditions, and on a good diet. After the experimental period, kill the rat, cut out the implanted apex with the surrounding soft tissue and prepare it for histologic examination. Examine the specimen for inflammation, resorption, new building of cementum or any unknown factors in the experiment, which will show whether the root canal treatment is satisfactory or not.

The judges will be men from both the medical and the dental profession.

It is not compulsory to use the biologic method. Contestants may prove that a method is satisfactory by any other method which is equal in value to the biologic method.

This is the last call. Enough has been said of good, best and worst root canal treatment. Proofs are needed.

This contest may show that there is one best method. In that event, no one has the right to use or teach other methods until he can prove that his is the better. It may show that we have many methods equally good; or it may show that no method used at present is satisfactory, and the search will have to continue.

The result of such a contest will be of the greatest value in promoting research. It encourages research before, and assures it afterwards.

It is true that the search for a better method will be never ended, but periods of investigation may be finished and new ones started.

This contest finishes the period of speaking without proof, and starts the period of proofs, which requires but little speaking.

The attention of those men who are particularly interested in the subject of root canal therapy is especially called to this contest. Nothing has been done thus far to prove the value of any methods. Clinically, most men show favorable results. But by some it is asserted that clinical results are not sufficient, and that all teeth with dead pulps must be extracted. Proof is needed from both sides.

For details, write to the secretary of the Commission for Scientific Research, Dr. I. Weinmann, Frank St. I., Vienna, Austria. IX.

If some one wishes to use the biologic method for proving his results, but has not the equipment for histologic technic, there are four laboratories, as the commission announces, which will be glad to give any help needed: Histologisches Laboratrium des Zahnarztlicher Institutes, Wahringer Strasse 25A. (Director, Dr. B. Gottlieb), Vienna IX. Austria; Zahnarztliches Institute der University Breslau Burgfeld 17-19, Breslau, Germany. (Director, Dr. H. Euler); Research Dept. of the Chicago College of Dental Surgery, 1747 West Harrison St., Chicago,

Ill. (Director, Dr. B. Orban), and the John Hampton Hale Research Laboratory of the Royal Dental Hospital and School of Dental Surgery, Leicester Square, London, England. (Director, Dr. E. W. Fish.)

Help or information is readily available. The competitors must be ready with their work by July 1, 1930.

A few words must be said about the prize. Dr. B. Gottlieb called the attention of the profession of the world to this competitive research one and a half years ago. He asked that every one who is interested in this problem, and we believe every dentist is, contribute to the prize, if only a few cents. The $1,000 prize is contributed by dentists from Europe.

This question has been discussed also by the Research Commission of the American Dental Association, without definite action being taken.

Research work needs individuals and not organizations. BALINT ORBAN.

CORRESPONDENCE

To the Editor:

Since reading the article that appeared in the September number of THE JOURNAL OF THE AMERICAN DENTAL ASSOCIATION, entitled "Women in Dentistry 1855-1880," I have wanted to write to those who prepared the article and compliment them on the thoroughness and the interesting manner with which they presented it. Although it deals with the period 1855-1880, there is one item I have that might be of particular interest to the compilers of the article.

Among the Decrees for the Regulation of the Dental Art prepared in France, there is one dated April 19, 1755, which had among other things forbidden women from the practice of dentistry. ". . . How would it be possible that women and girls, whom the decency of their sex excluded from the courses of anatomy and osteology can acquire a capability sufficient to practice some parts of surgery (truss-makers and dentists) with success, when, after many nights of hard labor, often the experience of the greatest masters is scarcely sufficient for those operations! . . ." Charles VIII, in 1484, had withdrawn from women the right to practice surgery; another edict of 1694 formulated the same ordinances.

I also find that among the list of dentists practicing in Paris in 1759 the names of Madame Calais, who was a student of Guraldy, practicing at Grenelle-Saint-Honoré. Her name appears as late as 1777. These is another, Madame Hervieu Rue Goffroy L'Asnier. These two are the earliest names of women that I have who have studied under preceptors and endeavored to practice dentistry as it was then constituted among the better men. There are several others, but they were mainly "toothe drawers."

B. W. WEINBERGER,
New York City.

NEWS

Section Officers, 1929 Session: The following have been appointed as officers of the scientific sections for the Seventy-First Annual Session of the American Dental Association: Operative Dentistry, Materia Medica and Therapeutics: chairman, Frank W. McDonald, New York City; vice chairman, H. B. McCarthy, Baltimore, Md.; secretary, Edouard M. Hall, Kansas City, Mo. Full Denture Prosthesis: chairman, E. B. Owen, St. Louis, Mo.; vice chairman, G. W. Holliday, Richmond, Va.; secretary, O. M. Dressen, Milwaukee, Wis. Partial Denture Prosthesis: chairman, William A. Squires, New York City; vice chairman, F. F. Whitcomb; secretary, Herman A. Maves, Minneapolis, Minn. Oral Surgery, Exodontia and Anesthesia: chairman, A. L. Frew, Dallas, Texas; vice chairman, Roy S. Hopkinson, Milwaukee, Wis.; secretary, Earle H. Thomas, Chicago, Ill. Periodontia: chairman, Clayton H. Gracey, Detroit, Mich.; vice chairman, Gillette Hayden, Columbus, Ohio; secretary, Robin Adair, Atlanta, Ga. Orthodontia: chairman, Charles R. Baker, Evanston, Ill.; vice chairman, Abram Hoffman, Buffalo, N. Y.; secretary, Max E. P. Ernst, St. Paul, Minn. Mouth Hygiene and Preventive Dentistry: chairman, Robin Adair, Atlanta, Ga.; vice chairman, George H. Wandel, Chicago, Ill.; secretary, E. F. Sullivan, Boston, Mass. Histology, Physiology, Pathology, Bacteriology and Chemistry (Research): chairman, Theodore B. von Beust, Louisville, Ky.; vice chairman, Russell W. Bunting, Ann Arbor, Mich.; secretary, Edward H. Hatton, Chicago, Ill.

Death of Professor Weiser: Word has just been received that Prof. Rudolph Weiser, of Vienna, Austria, died September 24, at the age of 70. Professor Weiser was the head of the Dental School of the University of Vienna and one of the leading men of Europe.

Dr. Weeks Heads Woman's Association: Dr. Haidee Weeks of New Orleans, La., is the newly elected president of the Association of American Women Dentists.

DEATHS

Bowe, John H., Oak Park, Ill.; Northwestern University Dental School, 1905; died, September 22, of cerebral hemorrhage; aged 47.

Carlson, Arthur C., Minneapolis, Minn.; University of Minnesota, College of Dentistry, 1916; died, August 30; aged 34.

Dutton, Lorenzo B., Roxbury, Mass.; died recently.

Flynn, Hiram A., Denver, Colo.; Temple University College of Dentistry, 1885; died, August 1; aged 72. Dr. Flynn was a past president of the state and local dental societies. He was connected for many years with the Colorado College of Dental Surgery in the capacity of teacher of anatomy, trustee and dean.

Goddard, James Watson, Ardmore, Pa.; Pennsylvania College of Dental Surgery, 1902; died, August 17, after an operation for stomach trouble.

Hassold, William Lewis, East Troy, Wis.; Marquette University School of Dentistry, 1906; died, August 20; aged 50.

Kiebler, R. C., Duncan, Okla.; University of Louisville School of Dentistry, 1908; died, June 29.

Kuhnmuench, Andrew J., Milwaukee, Wis.; University of Pennsylvania College of Dentistry, 1901; died suddenly, September 17; aged 48.

Lanning, George Richard, Sioux Falls, S. D.; Chicago College of Dental Surgery, 1899; died recently.

Leonard, Benjamin Fred, Plainfield, N. J.; University of Maryland, School of Dentistry and the Baltimore College of Dental Surgery, 1900; died, May 29.

McGinley, Wayne Lincoln, Newville, Pa.; University of Pittsburgh School of Dentistry, 1922; died, September 13; aged 31. Dr. McGinley was a veteran of the World War.

Merchant, P. H., Giddings, Texas; died, July 13, after a long illness with heart disease.

Owens, Edward B., Merrill, Wis.; died, September 16; aged 87.

Pairo, W. E., Washington, D. C.; George Washington University, Dental Department, 1899; died, September 22; aged 63.

Reisser, Otto H., Lakewood Ohio; Western Reserve University School of Dentistry, 1902; died, September 5.

Sargent, A. I., Galesburg, Ill.; Chicago College of Dental Surgery, 1896; died September 5; aged 58.

ANNOUNCEMENTS

CALENDAR OF MEETINGS*

NATIONAL SOCIETIES

American Dental Association, Washington, D. C.

STATE SOCIETIES

District of Columbia, at George Washington University, Washington, D. C., second and fourth Tuesdays in each month from October to June inclusive.

December (1928)

Ohio, at Toledo (4-6).

May (1929)

Iowa, at Cedar Rapids (7-9).
New York, at Rochester (15-17).
West Virginia, at White Sulphur Springs (20-22).

Chicago Dental Society Midwinter Clinic, Jan. 14-16, 1929.

Dental Protective Association of the United States, Chicago, Ill., Dec. 17, 1928.

First District Dental Society of the State of New York, Dec. 3-6, 1928.

MEETINGS OF STATE BOARDS OF DENTAL EXAMINERS

Connecticut, at Hartford, Nov. 20-22, 1928. Arthur B. Holmes, Recorder, 80 Central Ave., Waterbury.

*Announcements must be received by the fifth day of the month in order to be published in the forthcoming issue of THE JOURNAL.

Indiana, at Indianapolis, Nov. 12, 1928. Secretary-Treasurer, J. M. Hale, Mt. Vernon.

Iowa, at Iowa City, Dec. 10-13, 1928. State Department of Health, Capitol Bldg., Des Moines.

Kansas, at Wichita, first week in December. John L. Parkhurst, Secretary, Humboldt.

Massachusetts, at Boston, Oct. 9-11, 1928. Secretary, W. Henry Grant.

Michigan, at Ann Arbor, Nov. 19-24, 1928. Secretary, J. W. Lyons, 617-621 Dwight Bldg., Jackson.

Minnesota, at Minneapolis, Jan. 11, 1929. F. E. Cobb, Secretary.

New Jersey, at Trenton, Dec. 3-8, 1928. Secretary, John C. Forsyth, 148 West State St., Trenton.

North Dakota, at Grand Forks, Jan. 8-11, 1929. Gilbert Moskau, Secretary, Grand Forks.

Pennsylvania, at Philadelphia and Pittsburgh, Dec. 5-7, 1928. Secretary, Alexander D. Reynolds, 4630 Chester Ave., Philadelphia.

Rhode Island, at Providence, Dec. 11-13, 1928. Albert L. Midgley, Secretary, 1108 Union Trust Bldg., Providence.

DENTAL PROTECTIVE ASSOCIATION OF THE UNITED STATES

The annual meeting of the Dental Protective Association of the United States will be held at the Palmer House, State and Monroe streets, Chicago, Ill., December 17, at 4 p. m. The report of the officers will be given, a board of directors will be elected and such other business as should come before the association will be transacted. All members are urgently requested to be present.

By order of the Board of Directors:

J. G. Reid, *President*,
D. M. Gallie, *Vice Pres.-Treas.*,
E. W. Elliott, *Secretary*,
55 E. Washington St.,
Chicago, Ill.

CONNECTICUT DENTAL COMMISSION

The Connecticut Dental Commission will meet at Hartford, November 20-22, to examine applicants for license to practice dentistry and dental hygiene and to transact any other business proper to come before them. For further information, apply to

Arthur B. Holmes, *Recorder*,
80 Central Ave.,
Waterbury.

IOWA STATE BOARD OF DENTAL EXAMINERS

The Iowa State Board of Dental Examiners will meet at the State University of Iowa College of Dentistry, Iowa City, December 10-13, at 9 a. m., for the purpose of examining applicants for license to practice dentistry in Iowa. An examination for dental hygienists will also be given. All papers and credentials must be filed with the State Department of Health, Des Moines, at least fifteen days prior to the date of the examination. For further information and application blanks, address the State Department of Health, Capitol Building, Des Moines.

KANSAS STATE BOARD OF DENTAL EXAMINERS

The Kansas State Board of Dental Examiners will hold their examinations the first week in December at Wichita, provided there are enough applicants to justify a meeting. For further particulars, address

John L. Parkhurst, *Sec'y-Treas.*,
Humboldt.

STATE OF RHODE ISLAND BOARD OF REGISTRATION IN DENTISTRY

A meeting of the Rhode Island Board of Registration in Dentistry for the examination of candidates will be held at the State House, Providence, December 11-13, beginning each day at 9 a. m. Candidates shall present to the secretary of this board, at least one week before the examination, a written application on a form provided by the board, together with the examination fee of $20.

Albert L. Midgley, *Secretary*,
1108 Union Trust Bldg.,
Providence.

STATE OF MINNESOTA BOARD OF DENTAL EXAMINERS

The next meeting of the Minnesota State Board of Dental Examiners will be held at the College of Dentistry, University of Minnesota, Minneapolis, January 11. Applications must be in the office of the secretary by January 1.

F. E. Cobb, *Secretary*,
601 Donaldson Bldg.,
Minneapolis.

NORTH DAKOTA STATE BOARD OF DENTAL EXAMINERS

The North Dakota State Board of Dental Examiners will hold the next meeting at Grand Forks, January 8-11. Applications for examination must be in the hands of the secretary by December 29.

GILBERT MOSKAU, *Secretary,*
Grand Forks.

DENTAL SOCIETY OF THE STATE OF NEW YORK

The Dental Society of the State of New York will hold its sixty-first annual meeting at Rochester, May 15-17. A cordial invitation is extended to all ethical dentists who are members of state societies to attend. The society will also be pleased to extend a cordial welcome to all ethical Canadian dentists. For information with reference to the exhibits, write to E. G. Link, 226 Cutler Building, Rochester; Clinics, John T. McIntee, Chairman, Cutler Building, Rochester; Literary Exercises, etc., A. P. Burkhart, Secretary, 57 East Genesee Street, Auburn.

A. P. BURKHART, *Secretary.*

FIRST DISTRICT DENTAL SOCIETY STATE OF NEW YORK

The December Meeting for Better Dentistry of the First District Dental Society of New York will be held again this year at the Hotel Pennsylvania, December 3-6. This is the fourth of these meetings. The same plan of subscription will prevail this year, $5 admitting subscribers to all lectures, clinics, etc. The clinic attendance at these meetings is limited and subscriptions are registered in advance, giving the audience the opportunity to avail themselves of all the clinicians have to offer.

Among the lectures and clinicians who have accepted invitations are: A. D. Black, C. N. Johnson, H. B. Pinney, Chicago, Ill.; Sherman L. Davis, Bloomington, Ind.; Robert R. Gillis, Hammond, Ind.; Edward H. Jackson and P. C. Lowery, Detroit, Mich.; Carlos H. Schott, Cincinnati, Ohio; Edouard M. Hall, Kansas City, Mo.; Robert H. Ivy and Emerson R. Sausser, Philadelphia, Pa.; Percy R. Howe and Benjamin Tischler, Boston, Mass.; Raymond A. Albray and Thomas Asche, Newark, N. J., and the Logan Clinic Club of eight members, Portage, Pa.

An oral hygiene luncheon will be held Wednesday noon, at which time Percy R. Howe, President of the American Dental Association, will speak, as well as Harry B. Pinney, the Secretary. Other speakers will be John B. Watson and William Welch of Johns Hopkins University. Topic discussions will be held Thursday afternoon and the general clinics on Thursday evening.

The Manufacturers' Club of the Amerıean Dental Trade Association and the Independent Dental Manufacturers' Association will hold exhibits of the most recent appliances and materials in the hotel during this meeting.

Address all communications relative to subscriptions to Edith M. Davies, Executive Secretary, 2 East 103rd Street, New York City.

JOHN T. HANKS, *Chairman.*

PATENTS OF INTEREST TO DENTISTS

1527832. Toothbrush holder, Silas T. Bragg, Astoria, and A. R. Illing, Long Island City, N. Y.

1527845. Toothpick, Hal S. Daniel, Mexico, Mo.

1527853. Toothbrush, William Ferdon, Omaha, Nebr.

1527475. Combination dental chair and engine, J. P. Fletcher and F. P. Stone, Carlisle, Pa.

1527927. Apparatus for use in administering gases, vapors, and the like, H. W. C. L. Schroder, Lubeck, Germany.

1533605. Dental and surgical lamp, C. R. Pelton and H. W. Houghton, Detroit, Mich.

1533474. Dental casting device and process, John C. Shotton, Lakewood, Ohio.

1533625. Inlay vulcanizer and press, J. H. Williams, West Palm Beach, Fla.

1535017. Investing destructible patterns for inlay fillings, Jean Cline, Portland, Ore.

1534171. Dental floss-guiding means, Josiah B. Fickes, Omaha, Nebr.

1534952. Casting of dentures in metal, H. L. Hobday, and F. W. Nunn, Melbourne, Australia.

WILL A "PACKAGE LIBRARY" HELP YOU?

SOME PACKAGE LIBRARIES*

Compiled by and Available for Circulation from

THE LIBRARY BUREAU
American Dental Association
58 E. Washington St., Chicago, Ill.

Alveolectomy or surgical preparation of the mouth for dentures.
Amalgam fillings and physical properties of amalgam.
Apicoectomy.
Baked porcelain in dentistry.
Best time for orthodontic treatment.
Business side of dentistry.
Cast gold inlays.
Cavity preparation.
Children's dentistry.
Cleft lip and palate.
Comparative dental anatomy.
Condyle path movements.
Cysts.
Dental assistants.
Dental caries.
Dental cements.
Dental hygienists.
Diet and nutrition in relation to teeth.
Endocrine glands in relation to teeth.
Esthetics in denture construction.
Ethics.
Ethylene anesthesia.
Extraction of teeth.
Fixed and removable bridgework.
Focal infection.
Foreign bodies of dental origin in lungs and esophagus.
Fractures of the jaws.
Full denture construction.
Habits as factors in malocclusion.
Histologic studies of teeth.
History and progress of dentistry.
Hospital dental service.
Impression technic.
Indirect inlay technic.
Industrial dental service.
Interpretation of dental roentgenograms
Ionic medication.
Local anesthesia.
Maxillary sinus and antrum—diseases and treatment.
Muscle exercises and training in orthodontia.
Nitrous oxid-oxygen anesthesia.
Oral diagnosis.
Oral manifestations of systemic conditions.
Orthodontic appliances.
Osteomyelitis of the jaws.
Porcelain inlays.
Postoperative complications and care.
Preoperative examination of patients.
Preparation of cavities with hypersensitive dentin.
Procain (novocain) dermatitis.
Prophylactic odontotomy.
Pulp conservation including discussions and methods of pulp capping.
Root canal treatment.
School dental service.
Studies of saliva.
Surgical treatment of pyorrhea.
Toothbrushes, tooth pastes and mouth washes.
Traumatic occlusion.
Trigeminal neuralgia.
Tuberculosis and dentistry.
Ultraviolet ray in dentistry.
Use of elevators in extraction.
Vincent's infection.

*Each package library contains information on one of the subjects listed and is composed of reprints, magazine clippings, etc., on that subject only. The packages are loaned to members of the Association for a period of one week after date of receipt. The charge for borrowing a package library is 50 cents. No packages are sold. In response to requests, packages will be compiled on other subjects pertaining to dentistry.